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ENVIRONMENTAL SYSTEMS DIVISION

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222 S. RIVERSIDE PLAZA - SUITE 1870 CHICAGO, ILLINOIS 60606 (312) 648-0002 FAX (312) 648-0551

January 24, 1989

Dr. Winifred Oyen
Midland Health Department
125 W. Main Street
Midland, MI 48640

Dear Dr. Oyen:

Enclosed please find the following information for the Dow Midland Superfund Site located in Midland, Michigan. This Information Repository is provided you by the U.S. EPA through this office.

Transcripts from the April 28, 1988 public meeting

This repository should be available for public review during normal business hours.

According to the Superfund Amendments and Reauthorization Act of 1986 (SARA) 9617(d)

"...each item developed, received, published or made available under this section shall be available for public inspection and copying at or near the facility at issue."

Periodically, updated information will be sent to you by the U.S. EPA or this office. This information should be hole-punched and placed in the repository notebook. It is very important that all documents sent to you be placed in the repository upon receipt. Additionally, no extraneous documents (newspaper clippings, correspondence, etc.) may be kept in the repository.

If you have any questions or require additional information, please feel free to contact me at (312) 648-0002.

Sincerely,

Mary F. Blaney
Environmental Scientist

Enclosures

cc: J. Perrecone



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ENVIRONMENTAL SYSTEMS DIVISION

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January 24, 1989

Ms. Emilia Parker
Grace A. Dow Memorial Public Library
1710 W. St. Andrews Drive
Midland, MI 48640

Dear Ms. Parker:

Enclosed please find the following information for the Dow Midland Superfund Site located in Midland, Michigan. This Information Repository is provided you by the U.S. EPA through this office.

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If you have any questions or require additional information, please feel free to contact me at (312) 648-0002.

Sincerely,

A handwritten signature in cursive script that reads 'Mary F. Blaney'.

Mary F. Blaney
Environmental Scientist

Enclosures

cc: J. Perrecone



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ENVIRONMENTAL SYSTEMS DIVISION

222 S. RIVERSIDE PLAZA - SUITE 1870 CHICAGO, ILLINOIS 60606 (312) 648-0002 FAX (312) 648-0551

January 24, 1989

Mr. Kurt Shaffner
Ingersoll Township Hall
4400 Brooks Road
Midland, MI 48640

Dear Mr. Shaffner:

Enclosed please find the following information for the Dow Midland Superfund Site located in Midland, Michigan. This Information Repository is provided you by the U.S. EPA through this office.

Transcripts from the April 28, 1988 public meeting

Risk Management Recommendations for Dioxin Contamination at
Midland, Michigan Final Report

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According to the Superfund Amendments and Reauthorization Act of 1986 (SARA) 9617(d)

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Sincerely,

Mary F. Blaney
Environmental Scientist

Enclosures

cc: J. Perrecone



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Mr. John Perrecone
TES IV Primary Contact
U.S. Environmental Protection Agency
Region V
230 South Dearborn Street
Chicago, IL 60604

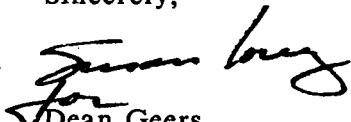
**RE: Contract No. 68-01-7351
Project No. 05-B177-00
Work Assignment No. 177
Dow/Midland Dioxin Site
Community Relations Support
CERCLA, Region V**

Dear Mr. Perrecone:

Please find herewith seven (7) copies of the Midland Risk Assessment. One copy has been sent to each of the three repositories.

If you have any questions or require more information, please feel free to call me at (312) 648-0002.

Sincerely,


Dean Geers
Regional Manager

Enclosures

cc: E. Howard, EPA Regional Contact
M. Blaney, Work Assignment Manager

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4-28-88

UNITED STATES
ENVIRONMENTAL PROTECTION AGENCY

IN the Matter of:
PUBLIC HEARING
RE: MIDLAND RISK ASSESSMENT

Transcript of the proceedings of the Public
Hearing held in the above entitled matter beginning on
Thursday, April 28, 1988 at or about 7:00 o'clock P.M. at
Northeast Intermediate School, 1305 East Sugnet, Midland,
Michigan, before Howard Zar, Project Manager, Dr. Mark
McClanahan, Dr. Ian Nisbet, Mr. Gary Amendola, Dr. Donald
Barnes, and Dr. J. Milton Clark.

1 MR. ZAR: Thank you. First of all I'd like
2 to thank the high school and the city also for helping
3 us set up the meeting.

4 This is a meeting on the dioxin risk assessment
5 and risk management for Midland. I'd like to make a
6 few brief remarks before introducing our speakers.
7 Tonight we're going to discuss the risk assessment and
8 risk management documents, also accept your comments on
9 the documents. There are copies back there at the back
10 of the podium or the back of the auditorium, rather,
11 also some fact sheets which also contain some advice on
12 how to file comments.

13 Just to review how we got here very briefly, in
14 1983 the Michigan DNR requested EPA with state
15 assistance, Michigan do a series of studies of dioxin
16 and other pollutants with much of the work being done
17 at the Dow Chemical plant. There's been a number of
18 reports published and several meetings on these studies
19 including wastewater, fish, ground water, surface
20 water, etcetera. These reports and findings have led
21 to a number of control activities by -- and efforts by
22 Dow Chemical in response to EPA and Michigan
23 requirements.

24 Also we've been using these results lately to
25 develop estimates of public health risks with the help

1 of a contractor, ICF Clements Associates. Two reports
2 that resulted from this effort, first is the "Risk
3 Assessment for Dioxin Contamination in Midland,
4 Michigan," that will be referred to tonight as the risk
5 assessment report. Second report is the Proposed Risk
6 Management Actions for Dioxin Contamination in Midland,
7 Michigan," that's a draft report, a public review draft
8 so-called, that will be referred to as the risk
9 management document tonight.

10 We want to advise you of the contents of these
11 reports tonight, listen to what you have to say about
12 them. There's an agenda that will tell you the
13 procedure, that's also in the back. You're -- As
14 indicated in the fact sheet we will be accepting
15 comments, written comments and comments made tonight.
16 Written comments may be submitted to us at any time up
17 until June 3rd. After that we will revise the risk
18 management document, we will provide it to people on
19 our mailing list, people who comment tonight and to
20 anybody who signs up in the back. You can see the
21 folks in the back to sign up for copies if you'd like.

22 Tonight we'll proceed as follows: EPA and its
23 consultant will brief you on the documents mentioned
24 then we'll accept statements from other government
25 officials. I understand the Michigan Department of

Public Health wishes to speak, also one of the councilmen from the city. Next we will accept questions and questions seeking clarification for the remarks that have been made. Only questions, however, at this stage. Then after the questions are finished we will accept formal comments. We have a stenographer here to record all the questions and statements, etcetera that have been made so we can review them carefully in preparing the final report.

I'd like to introduce briefly the panelists we have up here tonight. At my right Dr. Mark McClanahan is the toxicologist with the Emergency Response Branch of ATSDR, it's a federal agency that -- the ATSDR stands for Agency for Toxic Substances and Disease Registry, they're in Atlanta. He specializes in health assessments related to emergency response actions.

Next, second from the end on my right is Dr. Ian Nisbet, he's an independent consultant based in Massachusetts, specializing in exposure of risk assessments and toxic chemicals in the environment. He's consulted for the U.S. EPA on many occasions and is a recognized expert in the fields of dioxin, toxicology, exposure and risk. Dr. Nisbet was the senior author of the Midland risk assessment.

To my immediate right is Gary Amendola, who many

1 of you have seen before. He's an environmental
2 engineer with the U.S. EPA's field office in Cleveland.
3 He's been the project manager for our studies in
4 Midland for the last five years or more.

5 On my left, immediate left, is Dr. Donald Barnes,
6 who is chairman of U.S. EPA's Chlorinated Dioxin Work
7 Group and he was recently appointed director of U.S.
8 EPA's Science Advisory Board. Dr. Barnes is also one
9 of the authors of the Midland risk assessment.

10 Dr. J. Milton Clark on my far left is the
11 toxicologist with our pesticides and toxic substances
12 branch of the U.S. EPA in Chicago. And last myself,
13 I'm an environmental scientist with the U.S. EPA in
14 Chicago and I am chairman of our dioxin task force in
15 U.S. EPA office in Chicago.

16 We'd like to start, as I said, with a presentation
17 of the risk assessment and management reports. I'd
18 like to introduce Gary Amendola who will be followed by
19 Dr. Nisbet.

20 MR. AMENDOLA: Thank you, Howard. For my
21 presentation tonight or before I start my presentation
22 tonight I'd like to give you little bit of background
23 as to where we stand in this process that was initiated
24 in 1983 and how it folds in with EPA and state
25 environmental control programs.

1 As Howard indicated, many of the actions and
2 studies that we conducted over the last five years were
3 initiated in 1983 in response to a request from the
4 State Department of Natural Resources. In addition,
5 some of those programs and studies were conducted in
6 furtherance of our own investigative efforts. At this
7 time we are bringing these studies to a close. We have
8 completed all the -- just about all the field work and
9 the reports are out for public review and comment.

10 We believe that after June 3rd, the comment period
11 closing, and our issuance of a final risk management
12 report we will complete our special initiative of
13 dioxin contamination in Midland. At that time all
14 further work will be turned over to the various
15 regulatory programs in the state and in EPA, that will
16 include the RCRA permit, resource conservation and
17 recovery act, the NPDES wastewater permit and various
18 air permits and requirements. So we'll be very happy
19 to have the program reach that stage.

20 The focus of all of these investigations has been
21 dioxin or 2378-TCDD. I know that Don Barnes would
22 prefer it if -- that we use the term 2378-TCDD,
23 however, for purposes of this meeting tonight we will
24 be using the term dioxin. We did, however, investigate
25 many other toxic chemicals as well as dioxin in our

1 studies.

2 As probably most of you know in this audience
3 2378-TCDD or dioxin is an unwanted by-product of
4 certain chemical reactions. It has been found at
5 relatively high levels in certain types of waste
6 products from pesticide production and production of
7 other chlorinated organics, and more recently we're
8 beginning to find sources of dioxin that had not been
9 thought about earlier.

10 To give you a little bit of background about some
11 of the significant events that occurred with respect to
12 dioxin findings in Midland, Dow Chemical in 1978
13 notified the Department of Natural Resources of dioxin
14 contamination in fish collected from the river. At
15 that time the State Department of Public Health issued
16 a fish consumption advisory recommending that people
17 not eat any fish from the river.

18 From the period of 1978 to 1981 EPA cooperated
19 with the Department of Natural Resources in a number of
20 studies to try to determine the sources of dioxin to
21 the river. We certainly suspected the Dow Chemical
22 discharge and tried various different sampling programs
23 to find out how much or whether it was coming out at
24 that point. Most of those were not successful,
25 principally because we could not at that time analyze

the dioxin directly in the water at low levels.

In 1981 we initiated a fish bio-accumulation study where fish were suspended in cages in the plume from Dow's effluent, in the river upstream and downstream, and at various control sites. Those data plus an experimental large volume wastewater sampling program we initiated did indicate that Dow's effluent was a source of dioxin in the river.

Right about the time of release of that study there was a great controversy regarding dioxin contamination across the country and as a result of the findings here and elsewhere, the State Department of Natural Resources requested our assistance in doing further investigative work near Midland.

Following that work and the determination that Dow's effluent was a source, in 1984, the Michigan Water Resources Commission along with the Michigan Department of Natural Resources issued a final order of abatement to Dow for dioxin clean-up. And that order provided some interim effluent limitations that had to be met in the effluent, it gave Dow a period of time to construct an effluent treatment system and also required Dow to investigate many other possible control mechanisms within the plant.

With respect to the Michigan dioxin studies that

1 resulted from the state request, we initiated a soil
2 sampling program in 1983. Many of you may remember in
3 April of '85 we released the results of that study and
4 had a meeting, I believe it was right in this room as a
5 matter of fact. And in 1985 we released the results of
6 our drinking water studies. In 1986 the wastewater and
7 river studies were released. In 1987 we completed a
8 report on incinerator studies, which is being released.
9 And in 1988, where we are today, we've released a risk
10 assessment and our proposed risk management actions for
11 the dioxin contamination.

12 With respect to our soil studies, we had two
13 principal objectives, one is determine the levels of
14 2378-TCDD inside the Dow plant and in city soils and
15 try to come to some determination as to what were
16 possible sources. As part of that work we did a soil
17 sampling program in Midland, in Middletown, Ohio,
18 Henry, Illinois and four natural areas in Minnesota.
19 The purpose of selecting sites outside of Midland was
20 to find other industrialized and urban environments
21 that are similar but had different characteristics than
22 Midland, and also to find some background or natural
23 areas for comparative purposes.

24 The data from the soil study are summarized here.
25 Basically we did not find detectable quantities of

1 dioxin in any of the Minnesota natural areas. In
2 Henry, Illinois, in Middletown, Ohio only trace
3 quantities were found, barely above detectable levels
4 in only a few of the samples. In Midland, however, we
5 did find dioxin in soils from almost every -- just
6 about every soil sample tested. However, the levels we
7 found in Midland generally averaged well less than a
8 tenth of a part per billion. As characterized by this
9 graph the levels are well below the one part per
10 billion level that CDC has established as a level of
11 concern for dioxin in residential soils. Dioxin levels
12 at the edge of the Dow plant and inside the Dow plant
13 were significantly higher. Since that time, of course,
14 there have been many remedial actions taken to either
15 cover or cap the highest concentration areas inside the
16 Dow plant.

17 We also compared in 1985 the contamination found
18 inside of the Dow plant and in the City of Midland with
19 some of the other what are known as tier one and two
20 sites from EPA's national dioxin study. And generally
21 the levels inside the Dow plant were in the lower range
22 of what was found at other tier one and two sites and
23 levels outside the plant fortunately were also lower
24 than some found elsewhere.

25 Our conclusions were that the highest levels of

2378-TCDD were, of course, found within the Dow plant. The contamination in Midland averaged less than a tenth of a part per billion. And we suspected based upon the distribution of the dioxins in the soils and also with the evaluation of a considerable amount of work that Dow had completed at the same time that the air emissions were the likely source of dioxin outside the plant. It appeared from the nature of the distribution of the contamination inside the plant that some of it was due to fallout from air emission and some possibly due to process spills or other types of events of that nature.

We also conducted some drinking water studies. There was a concern that dioxin might be present in public drinking water supplies. We evaluated several -- Well, we evaluated three major public water supplies in Saginaw Bay, as well as several private and semi-public potable ground water wells in the area. We also wanted to determine, in addition to the dioxin, whether any other organic chemicals or other toxicants were present at levels that exceeded any drinking water criteria or standards.

This map sort of shows the location of the public drinking water supplies in Saginaw Bay. We took samples from the Saginaw-Midland intake, the Pinconning

intake and the Bay City intake. All of those samples turned out to be not detected at the lowest levels that could be achieved at that time. We also did not find dioxin in the private drinking water wells. There was an issue we had at the time of one of our contract laboratories had some contamination that required us to resample and go through some rather elaborate comparative testing to satisfy ourselves with the fact that we did not have dioxin in those wells, and that was our conclusion. We also did not find toxic organics present at levels that would exceed any of the drinking water criteria or recommended maximum contaminant levels established by EPA.

Also, as part of this work we did measure emissions from Dow Chemical's hazardous waste incinerator, and we wanted to compare the results of those emission tests with other incineration sources through the country. We did as part of that work some limited ambient air monitoring outside the Dow plant as well. Those samples were taken near the fence line of the plant and also out into the community.

This graph presents a comparison of Dow Chemical hazardous waste emissions as characterized by different air emission tests. The 1983 graph is a test that Dow had run. We had run the one in 1984, EPA did, and Dow

1 had run another one in 1987. So you can see that
2 there's been a significant reduction in emissions as
3 characterized by these tests.

4 The units presented there are dioxin equivalents.
5 Dr. Nisbet later in his talk will describe EPA's toxic
6 equivalents factors approach for the different families
7 of dioxins. And we converted these incinerator
8 emissions into grams per year here for comparative
9 purposes.

10 Now, we also have data from our national dioxin
11 study and we looked at various types of combustion
12 sources there as part of the tier four or combustion
13 source effort. And as you can see the Dow Chemical
14 results in 1983, '84 and '87 are highlighted. And the
15 emissions as characterized by the latest tests are
16 certainly well within the lower range of emissions from
17 all different types of sources. We have municipal
18 waste combusters, sewage sludge incinerators, other
19 hazardous waste incinerators and kraft recovery
20 boilers. There are also many other sources tested as
21 part of a national dioxin study that had emission rates
22 much lower than the lowest value showed here. Our
23 conclusions were fairly obvious, that the emissions
24 have decreased significantly since 1983.

25 We also found dioxin present in the ambient air

1 outside the plant. We tried to make some computer
2 modeling estimates of what the ambient concentrations
3 would be in the air outside of the plant based upon the
4 incinerator emissions, in other words, how much was
5 coming out of the stack. And we found from those
6 calculations that we could not account for the amount
7 of material outside of the plant measured in the air
8 with what was coming out of the stack. That plus the
9 data that we accumulated as part of our soil study led
10 us to believe -- leads us to believe that the amount of
11 dioxin in the incinerator currently is much less than
12 had been emitted in the past. Also, that there may
13 have been other sources contributing to levels in the
14 ambient air. These might include past process
15 emissions and wind blown dust from the plant site.

16 We also did some wastewater and continued fish
17 monitoring in the river. I might indicate that in the
18 fish monitoring there have been a series of native fish
19 studies in the Tittabawassee River from 1978 through as
20 late as 1987. And those studies were done to track the
21 levels in the fish. We also as part of Dow Chemical's
22 NPDES permit, the company is required to monitor
23 2378-TCDD in its effluent twice per month. So we've
24 been tracking those levels as well.

25 As you can see here this is a graph representing

the mass amount of 2378-TCDD from Dow's effluent going into the Tittabawassee River. There are two principal points I'd like to note here. First, the level of discharge has dropped significantly in November of 1985 and that was the time when Dow began operation of the mixed media effluent filter that was required by the Department of Natural Resources. And secondly there was another significant reduction beginning about July of last year when Dow began operating further controls for incinerator scrubber waters. But overall the trend here is very clear in that we have a situation where the dioxin levels going into the river have been reduced significantly.

Presented here is a graph showing the amounts of 2378-TCDD detected in game fish in the Tittabawassee River. Please note that in 1983 we have data for six fish, in 1985 it was a much larger study, we have data for 32 fish, in 1987 we have data for three fish. What I'd like to point out here that is significant is the average in '83 and '85 are very close together. And the types of fish analyzed here are principally Walleye. However, in 1985 it included Northern Pike, White Bass, small mouth Bass and a few other fish of that nature. In 1987 the limited data we had shows that the level of fish appears to be coming down. And

1 this is consistent with the reduction in discharge in
2 Dow's effluent.

3 We also have monitored Catfish and Carp in the
4 Tittabawassee River. And the bottom feeding fish tend
5 to collect or accumulate much higher levels of dioxin.
6 In 1983 in a cooperative program with the Department of
7 National Resources we analyzed 1 Catfish and 25 Carp
8 and had values ranging from about 10 parts per trillion
9 up to 530 with an average of about 50.

10 In 1985 we had three samples and these were
11 analyzed by Dow and I think the average there was 32.
12 So, there is some decrease noted, although the number
13 of fish are not significant in 1985 to draw a
14 conclusion. In 1987 again we have three fish with the
15 average dropping to six. The decrease in these fish,
16 it seems to be tracking back for the game fish
17 indicating that most probably the reduced discharge
18 levels from Dow are having a marked impact on the
19 fishery.

20 Our conclusions there are the average levels of
21 1983 and 1985 are about the same, particularly for the
22 game fish. The average levels in '87 appear to be
23 decreasing, but we only have limited data to make that
24 conclusion. And we're saying more studies are needed
25 in 1988 to confirm these more recent results. I might

point out we have collaborated with the Department of Health in Michigan and Department of Natural Resources on expanded fish monitoring program for 1988. In fact, the fish have already been collected and are at the analytical laboratories. Those fish will be analyzed -- most of them will be analyzed by the Department of Public Health for PCBs and other pesticides. Dow Chemical will be doing analyses of 2378-TCDD and U.S. EPA's research laboratory in Duluth, Minnesota will be doing some analysis of 2378-TCDD and for some of the other dioxins and furans that are present in fish.

With that I'd like to turn the program over to Dr. Ian Nisbet who will describe EPA's risk assessment approach and discuss our risk assessment results for Midland.

DR. NISBET: Risk assessment for a situation like this is a very complicated undertaking. A risk assessment document which is now being issued by EPA covers more 250 pages with more than 50 pages of tables. The summary which is made available by EPA, I believe copies are in this room, covers eight pages. I'm going to try to give you a little more information than is in the summary. I'm necessarily going to have to simplify the full risk assessment, but I will try to be as concise as I can.

1 What we try to do with this undertaking is to
2 estimate risk. Risk is the probability that someone is
3 going to be injured by a situation, in this case
4 exposure to dioxin in the environment of Midland.
5 We're not trying to measure injury. We're not trying
6 to go out and see who has been injured by exposure to
7 dioxin in the past. We are prospective, we try to
8 estimate the likelihood that people will be injured as
9 a result of present or future exposure. And when we
10 estimate that likelihood and determine the
11 circumstances which might lead to risk we want to do
12 something about it. This is an example of preventive
13 public health and it's difficult.

14 Risk assessment follows four standard components.
15 First, hazard identification. What does the chemical,
16 in this case dioxin, do? What kind of toxic effects
17 does it cause? Second, dose response evaluation. How
18 much do we have to be exposed to before we are likely
19 to be injured? Third, exposure evaluation. How much
20 are people exposed to? And finally, risk
21 characterization. Given the hazards, the dose response
22 information and the exposure evaluation what is the
23 likelihood that some people will be injured and how
24 many people? And then what can be done about it.

25 Let me go through these step by step. In hazard

1 identification we first of all review and analyze
2 toxicity data. Ideally we would like to study what
3 dioxin does in humans. Unfortunately, for various
4 reasons the direct information we have about what
5 dioxin does to people who are exposed to it is
6 inconclusive in various ways. The principal problem is
7 that although we have some information on people's
8 responses, physiological responses to exposure, we have
9 very little information about the actual magnitude of
10 that exposure. We have exactly zero dose response
11 information for people. Therefore, for risk assessment
12 we necessarily rely on animal data, controlled
13 experiments in which animals are exposed to dioxin in
14 laboratory.

15 Analyzing that evidence, which is being done
16 extensively by EPA, we weigh the evidence that the
17 substance, in this case dioxin, causes the various
18 toxic effects. That evidence is summarized in Chapter
19 2 of the risk assessment document. We then evaluate
20 whether the toxic effects which occur in one setting
21 will occur in other settings. And specifically what
22 that means is that we evaluate whether the toxic
23 effects that we observe in animals in the lab are
24 likely to occur in humans exposed usually at lower
25 levels. There are standard procedures for doing this

and we have followed those procedures in Chapter 2 of the risk assessment document.

EPA has identified three particular kinds of toxic effects as being most critical for risk assessment. By critical we mean these are the effects which occur at lowest doses in animals under experimental conditions and hence are the events most likely to occur in humans also exposed at low levels.

The first one of these is the increase in the risk of cancer. Dioxin increases the risk of cancer in animals exposed to it for long periods under laboratory conditions. There are some indications that it may do so in humans also. There are several suggestive studies but none of them is conclusive. EPA has a standard procedure for classifying carcinogens according to the weight of evidence. Dioxin on this categorization falls into Group B, it's referred to by EPA as a probable human carcinogen, and the basis of that is that it does increase the frequency of cancer in animals and may do so in humans, although there is no direct conclusive evidence whether it does or does not.

The next stage in evaluating the potential for dioxin to cause cancer is to look at the dose response data. For carcinogens we believe there is likely to be

no threshold dose for which there is no effect; that is, any dose is likely to give rise to some effect and at low doses the risk is approximately proportional to the dose. The relationship between risk and dose is known as the potency factor and it's so identified on this slide in terms of a risk per unit of dose. The unit of dose is expressed in picograms. A picogram is one trillionth of a gram. And that is related to the body weight of the person ingesting it in kilograms. And the dose is expressed in picograms per kilogram of body weight per day. Most people weigh between 50 and 100 kilograms, therefore, a dose of one picogram per kilogram per day is between 50 and 100 picograms per day.

According to the risk assessment procedure, if a person were to absorb that quantity of dioxin everyday throughout life his risk of -- his or her risk of getting cancer might be on the range of 1.6×10^{-4} to the minus 4, that is about 1 in 6,000. That is the basis for all subsequent cancer risk assessments in the risk assessment document.

In addition to cancer there are two other critical effects, critical toxic effects of dioxin, which have been observed in animals. One is an effect on reproduction. Dioxin interferes with reproduction and

causes birth defects in various species of animals and the dose responsive relationships for those effects are well documented.

At similar low dose levels it also causes toxic effects on the liver. Based on those studies EPA has derived a series of bench mark values. These are dose levels at which it can be estimated that people can be exposed for short or long periods without substantial risk of adverse effects. We end up calling them safe levels because we're never quite sure if something is absolutely safe, but we believe that these dose levels can be absorbed into the body for short or long periods with an ample margin of safety.

For long term exposures these bench mark is known as the reference dose or RFD, and based on the animal studies we estimate the appropriate dose for the RFD is about one picogram per kilogram per day. That's the long term exposure for months, years or lifetime. For shorter periods of exposure the bench mark dose is known as a health advisory, and for single dose exposures, that is a dose you might get on one day from eating a highly contaminated fish, the estimated dose for the health advisory is 300 picograms per kilogram per day or for intermediate exposures on the order of a week or two it's about 28 picograms per kilogram per

1 day. These are the bench mark doses that are used for
2 all risk assessment.

3 So much for Chapter 2 of the risk assessment
4 report. We now move to the exposure assessment, which
5 is covered in Chapter 3. We're concerned with three
6 primary routes of exposure, inhalation of vapors or
7 airborne particulates contaminated with dioxin,
8 ingestion, specifically ingestion of fish or
9 inadvertent ingestion of soil, particularly by
10 children, and contact with the skin. It turns out that
11 skin contact is not a significant route of exposure in
12 this context mostly because the potential for
13 absorption through the skin is quite low for this
14 chemical.

15 Exposure assessment is a very complicated
16 procedure and raises many issues. Chapter 3 of the
17 risk assessment report extends over 100 pages because
18 there are so many different factors that need to be
19 considered and discussed. Specifically we are trying
20 to estimate the extent and the frequency of human
21 exposure by each one of the three routes of exposure.
22 We want to estimate how much people are exposed to, how
23 often, over how long a period. We want to estimate the
24 number of people exposed and we want to estimate how
25 certain are our estimates and how variable the exposure

is.

In this case, as in nearly all cases of human exposure to environment chemicals, the exposure is very variable. Some people are much more highly exposed than others depending on their habits, where they live and what they do. So we want to characterize that range of variability in human exposure and we do it not by trying to estimate the exposure of everyone, but of trying to estimate the exposure of a typical individual or perhaps an average, if we could do that, and we also want to characterize the people who are most likely exposed in order to indicate who is most at risk and where remedial measures should be focused. We don't try to get the extreme high, we don't try to estimate the individual who had the greatest exposure, we try to estimate someone with -- near the upper end of the range to characterize a substantial number of people who are at highest risk.

In Chapter 3 we considered a series of exposures in our areas, we considered exposure of our air, inhalation of contaminated air, we considered exposure via soil, primarily ingestion of contaminated soil, and we've considered ingestion of contaminated fish from the Tittabawassee River. In each case we've considered two exposure scenarios, a higher exposure which

1 characterizes the upper end of the exposure
2 distribution, and a lower exposure which characterizes
3 somewhere in the middle. We didn't have enough
4 information to be confident in saying we calculated the
5 average exposure or the immediate exposure but we
6 believe that these numbers are somewhere in the middle
7 of the range of likely exposures at Midland.

8 In the case of air we considered two locations of
9 residents, the lower exposure is for a person living in
10 the middle of a residential area of Midland, about a
11 mile away from the plant, the higher exposure is a
12 hypothetical person living very close to the fence line
13 of the Dow facility on the downwind side. And each of
14 these were characterized in 1984 by actual sampling of
15 the ambient air.

16 For the soil the main reason for the difference
17 between higher and lower exposure is the behavior of
18 the individuals. The main source of exposure is by
19 children who play on the ground and get soil on their
20 hands and then put their hands in their mouths, that
21 kind of exposure has been reasonably well characterized
22 now and is known to be variable. We took numbers from
23 the literature including a review that we had done
24 ourselves. The lower exposure was somewhere in the
25 middle of the range of recorded values, the higher

exposure was near the upper end of the recorded values. And we also considered an extreme case, a child with what is known as pica. Pica is an unusual disorder in which children have an unusual craving to put objects in their mouths and swallow them. This applies not only to soil but also things like leaded paint.

For the fish consumptions we considered only people who eat fish from the Tittabawassee River. That it serves as a limited population, certainly some hundreds of people and probably some thousands of people. Within that group we considered a variety of fish consumption patterns based on documentation of how much fish people eat in other areas. They range from what we call the occasional consumer, who might eat fish from the Tittabawassee River about once a month, up to higher consumers at the upper end of the range, we considered it possible that some people might eat fish from the Tittabawassee River as much as three times a week, the maximum consumer being someone who would eat not only game fish but also bottom fish such as Catfish, which are known to be more highly contaminated. There is a wide range of possible intakes but they're all from within a limited population.

Within all of those scenarios, particularly the

1 higher exposure scenarios, we followed a standard
2 procedure of using what we call conservative exposure
3 assumptions. We don't know exactly how much fish
4 people eat or how much air they breath or how much soil
5 they eat. We took values from the literature, but in
6 doing so, to avoid underestimating risk we tended to
7 take the higher values. In particular, we used all
8 environmental data from 1983 to 1987. We averaged all
9 the fish data between 1983 and 1987, even though, as
10 you've just seen, there's some limited evidence that
11 the levels in fish have fallen substantially in 1987.
12 We don't know that for certain yet. We used soil data
13 from a 1983 survey and we used air data from a 1984
14 survey. It is quite possible that levels of exposure
15 have begun to go down. We don't know that yet, but we
16 have structured the risk assessment so that if the 1988
17 surveys indicate that the levels have continued to go
18 down then all the exposure and risk calculations can be
19 redone.

20 We also calculated exposure specifically for
21 people who are long term residents of Midland. For the
22 cancer risk assessments we essentially considered
23 people who are -- who will be lifetime residents of
24 Midland. For the air exposure we considered people who
25 were breathing the air at the specified location for 24

hours a day but not people who commuted outside. And in the case of the higher exposure scenarios we took all of the high intake rates deliberately to characterize the upper end of the exposure distribution. So in evaluating the risks you should be aware that we are considering the long term residents, we're considering current levels assuming that they will not go down and in the higher exposure scenarios we are deliberately looking at the upper end in order to characterize those highly exposed people.

Everything I've said up to now has been concerned with 2378-TCDD. In addition to 2378-TCDD there are a number of other dioxins and furans which have been detected in the Midland environment. 2378-TCDD is the -- probably the most toxic and the most characteristic of this -- these two families of chemicals which contain altogether 210 chemicals.

In order to characterize risks posed by exposure to complex mixtures, EPA has done an approach known as the toxicity equivalency factor approach. This is based on the scientific knowledge that most compounds in these two families act in the same way as 2378-TCDD only they are less potent. By less potent I mean that larger quantities of these other compounds are required to cause the same effects as the quantities of

2378-TCDD. So to estimate the toxicity of these individual compounds we estimated exposure to and multiplied them by a relative potency factor in order to come up to an equivalent form of the dioxin, 2378-TCDD. We then add up the effect of all the compounds and estimate the total amount of dioxin equivalents to which people are exposed. This procedure has now become standardized, it's reasonably well accepted in the scientific community and we have used it uniformly throughout this risk assessment where we have such data.

At Midland we found that 2378-TCDD was by far the most important single compound of these 210. In soil this one single compound contributed about 90 percent of the total toxic equivalents. In fish, however, it only contributed about 40 percent. That is, we estimate the total risks posed by the mixture to be about two and a half times those posed by 2378-TCDD itself. So it's important to consider those in fish but they are not overwhelmingly important. And as I say, 2378-TCDD is the most important single compound.

We've now covered the -- the first three components of risk assessment, hazard identification, the three critical toxic effects, dose response assessment, we identified three bench marks, exposure

assessment, we have exposure assessed for about twelve different exposure scenarios, and now we put these two together to estimate -- characterize the risks that would arise under each of the scenarios.

This slide characterizes our estimates, which are presented in Chapter 4 of the risk assessment document of the cancer risks which might result from exposure to dioxin due to the entire mixtures in Midland as a result of the exposure scenarios and under the rather conservative assumption which I've described. And it goes -- of uncertainties in both the exposure and the dose response evidence we have characterized these only to the nearest power of ten.

Working from the bottom of this table, from the bottom right, exposure via air and soil under the lower estimates, that's the somewhere in the middle of the range of exposure is likely to give rise to cancer risks, in the ballpark of one in a hundred thousand or one in a million. Those are not high numbers, those are ranges of cancer risks at which EPA usually begins to consider remedial actions. In soil and air the only exposure scenarios which lead to estimated risks greater than one in a hundred thousand are these two which are the extreme exposure assumptions, a person living close to the Dow fence line throughout life and

exposed 24 hours a day to current levels of airborne contamination or a child with pica who lives in the residential area of Midland throughout his childhood years. Each of these scenarios is pretty unlikely. They are extreme, they can't be ruled out, but generally we feel that for air and soil numbers in this ballpark are characteristic and those are the numbers of our risk assessment.

On the other hand, risks resulting from consumption of fish are much higher. Our occasional consumer was a person who eats a modest size fish meal, about a fourth a pound of fish from the Tittabawassee River about once a month. According to the assumptions we have made which assume that present levels of dioxin would be maintained for long periods into the future that person might suffer an excess cancer risk as high as one in ten thousand. Individuals with higher exposure could suffer much higher cancer risks. This high sports fisherman is a person that eats a larger meal, say half a pound three times a week over a long period, that person's risk might be as high as one in a thousand. The maximum consumer is a person with similar consumption but someone who eats the more contaminated bottom fish, that person's risk could be in the ballpark of 1 percent. Those are high risks and

1 those completely dominate the cancer risks posed by
2 exposure to dioxin among all the routes that we
3 considered.

4 I'm not presenting slides for the non-cancer
5 risks, the risks of reproductive effects, of birth
6 defects and of liver toxicity, those are in the risk
7 assessment document in Chapter 4. They essentially
8 fall into the same pattern as these. For air and soil
9 in almost all cases the estimated exposures are below
10 the bench mark levels at which we estimate that the
11 likelihood of adverse effects is very low. It's only
12 for the child with pica and individuals living near the
13 fence line who would be at substantial risk of any
14 adverse non-cancer effects.

15 On the other hand, considering the fish exposures,
16 these same individuals will also be at risk of
17 non-cancer effects; that is, a pregnant woman eating
18 fish even at this low rate would be taking in dioxin,
19 dioxin equivalents at a rate greater than the RFD and,
20 hence, would not have an adequate margin of safety.
21 And individuals with a higher exposure would be at
22 correspondingly higher risk.

23 So that is the output of our risk assessment
24 exposure via air and soil is at -- risks resulting from
25 exposure via air and soil are at worst marginal. Under

1 extreme exposure conditions there may be some risk,
2 although these extreme exposure conditions are quite
3 unlikely. Exposure via fish make -- under the exposure
4 assumption, more conservative exposure assumption we
5 discussed in the report may lead to substantial risks
6 both of cancer and of non-cancer adverse effects.

7 That is the outcome of the risk assessment portion
8 of our study. The risk characterization of that leads
9 right on into the risk management area. I turn this
10 back to Gary Amendola.

11 MR. AMENDOLA: Thank you, Dr. Nisbet. As you
12 can see from the intertwined circles here the risk
13 assessment and risk management flow into one another.
14 The risk characterization leads to some sort of a
15 regulatory decision, and as part of that there is
16 consideration of much more than the risk assessment
17 results. In many cases there are various control
18 options that are considered as well as non-risk
19 analyses. They might be things such as statutory
20 requirements that would mandate a control option,
21 whether or not the control option resulted in a
22 significant risk reduction or they might be public
23 concerns or information about cost and economic
24 benefits.

25 Some of you may be wondering why we're having this

meeting here today in 1988 when a lot of this work was done in 1985. I think that's a very legitimate question. One of the reasons is this is, as Dr. Nisbet pointed out, a very complex task. And while this was going on there have been a lot of activities to minimize exposures and minimize emissions from Dow Chemical. The State of Michigan, for example, in 1984 issued its dioxin order we discussed earlier. In 1985 when we found high contamination -- high dioxin contamination on the site, a CERCLA or Superfund abatement order was issued to Dow for clean up of those sites. More recently in 1986 the state implemented a death suppression program with Dow. And we have ongoing record of RCRA permitting and NPDES wastewater permitting activities going on at the same time.

Also, I'd like to point out that there have been a number of actions that Dow has undertaken unilaterally to minimize some of these emissions and discharges that we've just spoken about. In the late 1970s most chlorinated phenols -- production of most chlorinated phenols was terminated at the plant site. Dow has installed a riverbank preventive system to collect contaminated ground waters at the site for treatment, and they have also approved their incinerator operations and otherwise complied with the permits and

orders issued by EPA.

So essentially upon evaluating the progress that's been made, the results that we're beginning to see in downward trends in environmental levels, when we got to risk management, you know, the obvious focus, of course, is looking at the sources, we found and concluded that many or most of the control options that need to be considered have either been installed or are being implemented. We were not faced with evaluating twelve different kinds of treatment for dioxin from Dow's incinerator or the wastewater discharge. Fortunately, we're in the situation of basically trying to monitor where we stand now and looking for further improvements where possible.

Accordingly we've developed risk management actions for Dow that focus on the wastewater and air. On wastewater as part of the next NPDES permit for Dow we've advised the state and have agreed with the state that conditions should be put in Dow's permit that should focus on research to determine whether any additional treatment of the Dow effluent is feasible. Second, we've asked for some studies as to the effectiveness of the incinerator treatment system and also on whether any sediments in Dow's tertiary pond would pass through the effluent filter and may be

□
1 contributing to the effluent discharge.

2 On air, Dow has been implementing a program of
3 improving its incinerator combustion conditions and
4 operating controls and we recommend that program
5 continue. Also, we recommend that Dow continue to
6 implement the dust suppression program to minimize wind
7 blown dust from the plant site. That program, again,
8 is a requirement of the MDNR.

9 Also, in our proposed risk management actions
10 we've outlined a number of monitoring programs that we
11 think might be appropriate. And again, these are all
12 things and points that we are seeking public comment
13 on. We believe it's appropriate that Dow Chemical
14 continue to monitor the wastewater discharge in
15 accordance with its permit condition so we can track
16 the levels. Some of you may be aware that the
17 Department of Natural Resources has indicated that the
18 desired level of dioxin discharged in Dow's effluent
19 should be about .1, 0.1 parts per quadrillion, which is
20 less than the current discharge rate of 1 to 2 parts
21 per quadrillion, and also much less than the current
22 discharge limit of 10 parts per quadrillion.

23 We recommend that there be some supplemental
24 incinerator emission and ambient air testing as part of
25 Dow's RCRA permit requirements, that Dow will have to

conduct additional incinerator trial burns to determine the destruction of efficiency of the incinerator. We recommend that some additional samples be collected at that time for specific purposes. Also, we're interested in having some ambient air testing done again to determine the effectiveness of some of these programs. We have one set of data collected in 1984 which was before any of the dust suppression programs or CERCLA remedial actions at the site were implemented.

Finally, we think that there should be continued Tittabawassee River fish monitoring. We're not finally but third. We have, in fact, as I indicated earlier, collaborated with the state and Dow on a program for 1988 to better characterize the levels in the fish. We also believe that it might be a good idea to check the river sediments, there's been a big flood here a couple years ago which probably moved and disturbed some sediments and we think there should be a program to evaluate sediment quality. And finally we think it might be appropriate to look at some limited food chain studies. For example, some of the aquatic life and birds and animals that live near the river. Also, to characterize some possible other human routes of exposure or possible routes of exposure, some limited

1 dairy sampling might be appropriate.

2 Okay. As part of our effort, as Dr. Nisbet
3 pointed out, we characterized the risk of consuming
4 Tittabawassee River fish as presenting the highest
5 exposure and also the highest risk to people who eat
6 those fish. Currently the MDPH or when the slide was
7 presented, in any event, the MDPH had an advisory
8 against eating Catfish and Carp. More recently they've
9 amended that to include game fish restrictions for
10 women of child bearing age, and I'm sure the Department
11 of Health will discuss that in detail later. We
12 recommend that people heed those advisories. Also, if
13 people are going to eat fish from the Tittabawassee
14 River, the state guidelines for cleaning the fish
15 should be followed. Those guidelines were designed
16 with the idea of trimming away those portions of the
17 fish that contain the most contaminants. And the risk
18 assessment document as well as the State Michigan
19 Fishing Guide describes in detail those procedures.

20 Finally, we felt it might be appropriate to make
21 some recommended actions for precautionary measures
22 regarding contaminated soils. And this type of
23 guidance or advice would apply to whether people were
24 living in Midland, Kalamazoo or New Haven, Connecticut.
25 We think that generally children -- parents who have

1 children with pica and parents of toddlers should
2 encourage children to try to keep soil out of their
3 mouths. That certainly is probably a lot easier said
4 than done.

5 Other common sense measures include thoroughly
6 washing your hands after exposure to the soil and
7 washing or peeling any homegrown vegetables you may
8 grow. We did collect some samples of homegrown
9 vegetables in Michigan -- or Midland rather, and we
10 don't have the final results yet, however, the
11 preliminary data tend to indicate that the root crops
12 such as beets and carrots do not absorb dioxin and
13 simple peeling to remove the contaminated soil would
14 probably remove any contamination.

15 That concludes our presentation. Right now I'd
16 like to turn the program back over to Howard Zar.
17 Thank you.

18 MR. ZAR: Thanks very much. We'll now take
19 statements from government officials. I believe Mr.
20 Lawrence Chadzynski from the Michigan Department of
21 Public Health would like to make a statement. I can
22 pass the microphone down to you at this point.

23 MR. CHADZYNSKI: Good evening, ladies and
24 gentlemen. It's a pleasure for me to be here, to drive
25 into the beautiful City of Midland. It was interesting

1 as we drove in we noted a sign as we were coming for
2 dinner and it said fresh Tittabawassee fish, all you
3 can eat. And we partook, it was delicious.

4 (Inaudible comments regarding EPA dinner
5 invitation from MDNR were made.)

6 This is a three part presentation. I've asked
7 that Dr. Benjamin Johnson, our staff epidemiologist and
8 physician, talk about the medical aspects of the
9 report. And Mr. John Hesse, marine biologist on our
10 staff, who will discuss the fish advisory and his
11 knowledge of fish.

12 In 1983 Howard Tanner, the then director of
13 Michigan Department of Natural Resources, submitted two
14 proposals to the United States Environmental Protection
15 Agency requesting federal support to conduct dioxin
16 studies in Michigan. Moreover, that these studies
17 should be part of a larger national effort designed to
18 answer the many questions related to dioxin
19 contamination. Ten years have elapsed since the
20 Michigan Department of Public Health first issued a
21 formal fish consumption advisory for the Tittabawassee
22 River.

23 In 1986 the center's fish advisory continued to
24 recommend against the eating of Carp and Catfish, but
25 reviewed the reduced levels of dioxin found in fish --

1 in the latest round of fish tested, removed the
2 limitation of eating game fish. A week ago we issued a
3 precautionary alert.

4 The EPA report before you today represents five
5 years of concerted effort and study. The report augers
6 well for Midland. The report shows that dioxin levels
7 in soil, air emissions and discharge waters are fine,
8 and that a downward trend is noted for fish sampling
9 during the course of the study. We commend EPA for
10 producing this excellent and comprehensive report. It
11 was quite an effort.

12 I would now like to briefly comment on the risk
13 assessment process presented in this report.
14 Nationally, and even within EPA, there appears to be
15 considerable debate of the risk assessment process in
16 both sectors, public and private. Barry Commoner in a
17 speech delivered to EPA in January of this year
18 commented on what is now referred to as Factor 16; that
19 is, that EPA was developing a new rationale for dioxin
20 cancer risk assessment that shows the risk is 16 times
21 lower than estimated by EPA in 1985. Lee Thomas, EPA's
22 administrator, recently estimated that he would spend
23 ten million dollars in fiscal year '89 on projects to
24 reduce the uncertainties and risk assessment to improve
25 our decision making responsibilities. We think that's

an excellent step.

The director of EPA's Office of Technology, Transfer and Regulatory Support quoted EPA plans to re-examine the assumptions that otherwise risk assessment. Part of this report states is the fact that the agencies all use different assumptions and arrive at different results. Examples of some of the changes the agency is considering is one, switching from the current assumption that people are exposed to a chemical 24 hours a day per day for several years to assuming an exposure of 16 hours a day for 10 to 35 years. Second, to consider only malignant tumors, not benign ones. And third, averaging the chemical's potency from experiments on a variety of animals instead of using a number derived on one single animal and the most sensitive one.

The report states that EPA's current approach is thought to overestimate human risk by a factor of ten. From our perspective in view of the literature and our own risk assessment process one observation can be made with some certainty, risk assessment provides us an important mathematical tool that substantially contributes to the decision making process, however, a risk assessment process is still in the early stages of development. There is much room for improvement but by

1 way of elimination of sources of uncertainty and of err
2 and recognizing the great amount of professional
3 judgment is still necessary. Dr. Vernon Houck,
4 director of the Center of Environmental Health stated
5 in the absence of other more certain data risk
6 assessment is all there is. Just as it should not be
7 denigrated as unhelpful because of its inevitable
8 limitations neither should it be oversold as passe. We
9 must apply the soundest professional and scientific
10 judgment available in order to shape up the policy
11 that's scientifically the best one.

12 In closing we would also like to commend Dow for
13 its efforts and cooperation with the regulatory
14 agencies in eliminating the dioxin problems in Midland.
15 We must continue to work together at the local, state
16 and federal levels. The making of the committment to
17 restoring the quality of our environment, the making of
18 our state for ourselves and for our product, I can
19 assure you that Governor Blanchard and Raj Wiener, the
20 Director of Public Health, share this commitment.
21 Thank you.

22 MR. ZAR: Dr. Johnson is next.

23 DR. JOHNSON: I, too, will sit down, if you
24 don't mind.

25 I'm Ben Johnson, I'm a physician and an

epidemiologist. I work with the State Health Department and Center for Environmental Sciences. I'd like to think being the only physician to speak brings us back to the health issues and just what we do know and what we suspect about the dangers of this chemical. And I'll challenge just for the moment with one thing. After millions of dollars of expenditure on investigations of this type there has yet to be a proven case of anything outside of chloracne in a human being.

Now, I'm not saying that there is no risk or that there cannot be, but we keep talking about cancer, we keep talking about liver disease, we talk about reproductive effects and we have yet to find any. And that we must continue looking because obviously in animal experiments and in laboratory experiments there have been good indications that this is a dangerous chemical, I don't doubt that. But I'd like to say as a physician I think first of the individual patients but I as an epidemiologist, as a scientist, we must look at broad populations. And that is what I think is sorely needed here.

We, as Mr. Chadzynski was saying, have a lot of risk estimation data based upon laboratory and animal experiments and then computer generated numbers that

1 may or may not mean anything. I have grave
2 reservations about them. What we need and sorely lack
3 is good human studies. These are not easy to come by
4 because in the first place, as Dr. Nisbet said, it's
5 very difficult to get dose response and a good estimate
6 of how much dose is given, we can't experiment with
7 human people with a dangerous chemical. But we do have
8 a number of them that result from worker studies, from
9 accidental exposures, and they're not as bad, I think,
10 as we've been led to believe.

11 We tend to put off the record the experience and
12 looking at the animals. For example, we have the very
13 excellent study that was done here on the Dow Chemical
14 workers. This covered many years and many thousands of
15 people. The results of that were essentially negative
16 in terms of cancer, birth defects or acne. The
17 original study where there was noted severe exposure in
18 workers was in Nitro, West Virginia. These people had
19 severe exposure. All of them in the study group had
20 chloracne. Chloracne indicates you've really got a
21 dose of it. The experience following these people over
22 the years has been up until now virtually negative,
23 nothing.

24 Also there are the recent Seveso, Italy study
25 where there was a large exposure accidentally, an

explosion, and many thousands of people were exposed. This was an opportunity to really look at the picture of whether there are reproductive effects. That study, which was recently released a month ago, showed us that in many, many thousands of births in the most exposed areas there were no unusual numbers of birth defects. That's very re-assuring. It's not absolute, not to say that it can't happen, but I think it's an important study.

As of two days ago the paper given on Times Beach, I think you're all familiar with that, this town in Virginia that was so heavily exposed -- or Virginia, Missouri that was so heavily exposed that they bought up the town and moved it away. But they studied those people very carefully, again, for reproductive effects and birth defects, nothing.

So these are important milestones and personally I believe human studies far more than the animals ones if I have a choice. Unfortunately, in a lot of cases we don't and we must go with the best information we have, either in the laboratory or in animals. So my feeling, especially with regard to reproductive effect, is that if there is a risk it is a very small one.

MS. HEBERT: Are you going to finish soon so we can talk?

1 DR. JOHNSON: Sure, in about two minutes.
2 Let me just finish up.

3 UNIDENTIFIED CITIZEN: Take all the time you
4 need.

5 MR. ZAR: The procedure is that we're going
6 to take comments from government officials then we'll
7 take questions and then statements. I don't think
8 there are too many --

9 MS. HEBERT: What do you mean government
10 officials? What do you mean?

11 MR. ZAR: Well, there's another gentleman
12 from the Department of Public Health that's asked to
13 speak and another gentleman from the city.

14 MS. HEBERT: So, what's --

15 MR. ZAR: Can you wait a few minutes --

16 MS. HEBERT: No.

17 MR. ZAR: We'll get to you, I'm sure.

18 MS. HEBERT: How does the hierarchy work
19 here?

20 DR. JOHNSON: I'll be very brief. So what I
21 wanted to say is with regard to pregnant women I
22 certainly don't minimize the risk, and as a physician I
23 know that we must do everything we can to protect them.
24 But I do know that we live in a dangerous world. There
25 are chemicals and there are exposures, there are toxins

1 and there are carcinogens in the very food we eat. And
2 if we want to protect everyone to the greatest extent
3 we'd have to put them in a glass case and feed them a
4 formula, which we don't know how to prepare because we
5 don't know what yet is dangerous.

6 So, we have to use a little common sense, I say
7 that we do the best we can to protect our people. And,
8 again, for that reason I agree, we should -- I go along
9 with the recommendation that even though there's very
10 little dioxin in all likelihood in fish now that for a
11 woman to be absolutely sure I would not have a lot of
12 fish any more than I would have any other kind of
13 dietary imbalances, a good general diet in pregnancy is
14 important. There are some known causes to pregnancy
15 abnormalities, number one is smoking, number two is
16 alcohol and somewhere way, way down the list is eating
17 fish out of the Tittabawassee.

18 MR. ZAR: The last speaker is from the
19 Department of Public Health is John Hesse.

20 MR. HESSE: Perhaps I can clarify some of the
21 information you've been hearing this past week and some
22 of which has been accurate and some hasn't about what
23 our recommendations are on eating the fish. The EPA is
24 entirely right that we're continuing our recommendation
25 not to eat the Carp and Catfish from the river. No one

1 should be eating those species, they're much more
2 heavily contaminated than the game fish.

3 With regard to game fish, we have a copy of our
4 press release last year in the back of the room that
5 talks about the recommendation as a precautionary
6 measure at this point until the situation be better
7 evaluated for women of child bearing age and pregnant
8 women not to eat more than one meal of game fish from
9 the river a month. That rate of consumption is
10 essentially equivalent to the one picogram per kilogram
11 body weight per day that Dr. Nisbet was talking about
12 as long term safe level, or I guess that's reasonably
13 close to what you call a reference dose.

14 We have been asked why aren't we saying or
15 recognizing these extreme risks that EPA's report
16 presents, and I think you heard tonight some of the
17 uncertainty factors. Dr. Nisbet very well covered some
18 of those and we take these factors into consideration
19 along with some of the other things that Dr. Johnson
20 mentioned as well in drawing our conclusions.
21 Quantitative risk assessments are a tool and we use
22 them to look at relative risks, one -- fish from one
23 body of water to another, but we -- extrapolation to
24 actual quantitative risks to humans have a great deal
25 of uncertainty associated with it and sometimes this

uncertainty factor is recognized by EPA that there can be several orders of magnitude for true risk and we feel that is often the case.

Other factors that we considered in our recommendation is that we do have some very current local information in terms of fish consumption, rates for anglers on the Tittabawassee River. This data was generated under contract by our department by Dr. Brad Smith, one of your local researchers from Delta College. He's here tonight and perhaps can answer questions for some of you afterwards. We will have copies of that report available if you write to our department and ask for it, we have to get copies made yet.

But the consumption rate that we -- he found from interviewing 703 anglers last summer showed that the 90 percentile or 9 out of 10 people fishing the river were not eating fish more than one meal per month. Actually, 44 percent of the fishermen were throwing them back. That nine that ate that one meal per month rate is essentially equivalent to the occasional consumer that Dr. Nisbet showed on the slide. And that in terms of the game fish concentrations is close to the rate of that reference dose of one picogram per kilogram per day.

1 So, there's just a small percentage of the
2 population, 10 percent, that may be eating more than
3 that. And what our advisory is trying to target are
4 those perhaps more sensitive proportions of the
5 population of women of child bearing age and pregnant
6 women in terms of protecting the possible effects of
7 the fetus, but at this point that is not very certain
8 of a risk.

9 The EPA report, of course, has pointed out
10 possible decline in concentrations in the fish, and
11 that this is probably going to be continuing as
12 discharges continued to decline. We've seen these
13 similar decreases in contaminants and in fish with
14 situations like when we brought DDT under control in
15 Michigan by banning it in 1969. PCB regulations went
16 into effect, we've seen 90 percent decrease in PCB
17 following the control of that. Mercury in the Lake St.
18 Clair area dropped within two years, started to show
19 dramatic decline as the fish here apparently are
20 starting to show and it's continued to decline.

21 The cancer risk numbers are -- remember that
22 they're generated using an assumption of 70 year
23 exposure at the rate that they calculated as an average
24 from 1983 to 1987, average concentration. And we can
25 very fairly safely assume that those concentrations

1 aren't going to continue for 70 years.

2 And another mitigating factor is the fact that the
3 risk assessments are done on uncooked, skin-on fillets.
4 And if people follow the recommendations of taking the
5 skin and the fat off, which we recommend, and it's in
6 our Michigan Fishing Guide and there are copies of that
7 back in the back room with our -- printed right in the
8 documents that's given to anglers, by removing that fat
9 and cooking it in ways to allow the fat to drip away
10 further reductions as high as 90 percent of some of the
11 other chemicals in the study that we funded in this
12 past year on dioxin reduction in Carp from the Saginaw
13 Bay. And this is brand new data, researchers at MSU
14 showed 40 to 70 percent reduction just by cooking the
15 fish by charbroiling. These are restructured Carp
16 fillets that are prepared in a certain way. And also
17 the fact that the human studies haven't shown evidence
18 that people are as sensitive as animals to this
19 chemical, perhaps they're much less sensitive.

20 The EPA policy change potentially coming up in the
21 next few months that we suggest that maybe dioxin
22 potencies aren't as great as they once thought is also
23 another factor. So those are some of the things that
24 we considered in our advisories. And I thought we
25 should reiterate the position, apart from Catfish,

1 there are a few people as shown in Dr. Smith's survey
2 that continue to eat this Carp and Catfish and there
3 probably always will be those people who ignore the
4 advisory, but we want to emphasize that they should be
5 avoided and your spreading that word would be helpful
6 to us.

7 Game fish are much lower than even most regulatory
8 numbers or most other states or any other agency has
9 set for a maximum level. Michigan uses ten parts per
10 trillion in terms of trigger level. Those game fish
11 are in the neighborhood of one part per trillion in
12 those 1987 Walleye and using the toxic equivalent
13 approach that brings them up to about four and a half
14 parts per trillion. If we can confirm that's true it
15 remains under our ten part per trillion trigger level.
16 State of New York uses 10 parts per trillion, Canada
17 uses 20 parts per trillion, FDA has stated a concern
18 level of 25 parts per trillion. So we feel these are
19 quite low levels and we'll continue to watch it and if
20 the levels appear to change higher than we previously
21 thought then, of course, we'll reconsider our position.

22 MR. ZAR: Thank you. I know we have one
23 request to speak from Mr. McCaffrey, City of Midland.

24 MR. McCAFFREY: I will not sit down. I don't
25 have any scientific data to give you. On behalf of the

1 citizens of the City of Midland, our Mayor regrets that
2 he could not be here this evening, he's down at a
3 sister city meeting, Midland, Alabama. And as the
4 Mayor pro tem I would like on behalf of the citizens of
5 this community to publicly thank the Environmental
6 Protection Agency, the Michigan Department of Public
7 Health and the Michigan Department of Natural Resources
8 for the fine upstanding efforts they have given this
9 community. They have given them the results of some,
10 from what I have seen, some brilliant scientific minds
11 at work, the cooperative effort between three agencies
12 and also the cooperative effort of the local industries
13 who have gone that extra mile, that extra step, which
14 is always required in a community that is concerned and
15 is aware. We thank you very much.

16 MR. ZAR: We thank you. Are there any other
17 officials that would like to speak?

18 (No response.)

19 MR. ZAR: If not we'd like to accept
20 questions of the panel and Department of Public Health,
21 if they're willing, on anything that's been said so
22 far. Just questions now, requests for clarification.
23 We'll take comments later.

24 MS. HEBERT: Yes.

25 MR. ZAR: Is this a question, ma'am?

1 MS. HEBERT: Yes, a question. About the
2 sample you took at the Rockwell dump when you were at
3 your Minneapolis lab and it looked like tea, and you
4 said to Larry Fink, if you tell anybody about this
5 you're going to be in deep trouble. Tell me about
6 that, huh? Tell me about that.

7 MR. ZAR: Is that me personally?

8 MS. HEBERT: You personally.

9 MR. ZAR: I never said such a thing.

10 MS. HEBERT: Yes, you did.

11 MR. ZAR: Are there any other questions?

12 MS. HEBERT: No, you don't ignore me, you
13 talk about it.

14 MR. ZAR: I remember no such thing. I'll be
15 happy to call Mr. Fink in the morning and ask him.

16 MS. HEBERT: Of course you do. And what
17 about the unknown unidentified hydrocarbons in all of
18 the water samples that we haven't yet identified, what
19 about those?

20 MR. ZAR: I'll answer that second question.
21 Mr. Amendola, do you want to try that one?

22 MS. HEBERT: Yeah, try that, Gary.

23 MR. AMENDOLA: Thank you, Howard. As part of
24 the study of some of the drinking water wells we
25 subjected the water samples to what are commonly

referred to as broad scan organic chemical analyses. And we, in our effort to try to be as complete as we could, we instructed the laboratories to identify all peaks that come out of these analyses to the extent that they can. And as part of the analytical work, the analysts, the consulting laboratories did an extensive analysis of these chromatograms and did identify several organic chemicals, many of which are common to oil and gas field type areas of which some of the Midland water samples are. Also, as part of that work there were some compounds that showed peaks that could not be identified. And we did not go back and redo analysis or try to figure that out because the initial effort was one that we believed was about as far as you could go without spending an exorbitant amount of money on each individual sample. We concluded from the analysis of the data we had that the levels of contaminants identified were certainly well below the -- either the drinking water criteria or standards or any maximum contaminant levels.

MS. HEBERT: 50 parts per trillion is kind of significant, Gary.

MR. AMENDOLA: Well, a peak that could show up in some of those analyses are not necessarily a toxic compound.

1 MS. HEBERT: You should have gone back and
2 try to identify the peaks, right?

3 MR. AMENDOLA: We attempted to identify the
4 peaks in the first analyses to the extent possible.
5 There is a limit to how far you can go in that type of
6 work and we think we did it.

7 MS. HEBERT: How's that? I don't understand
8 what you're saying.

9 MR. ZAR: Am I correct that this questioner
10 is Diane Hebert, representative of Greenpeace?

11 MS. HEBERT: I just live here, okay.

12 MR. ZAR: A resident of Midland, then?

13 MS. HEBERT: Yeah.

14 DR. OYEN: I'm Mrs. Oyen, a resident of
15 Midland from the local health department.

16 MR. ZAR: Would you like to come up?

17 DR. OYEN: No, I was just going to ask Dr.
18 Nisbet a question. You've been talking about one in
19 ten thousand increased risk of cancer. Would you put
20 it in perspective for people by mentioning what is the
21 risk to any of us from just existing and perhaps what
22 is the increased risk from smoking a pack of cigarettes
23 a day.

24 DR. NISBET: The average person in this room
25 has about 30 percent chance of developing cancer over

1 his or her lifetime and about 25 percent chance of
2 dying of cancer. That's about 2,500 chances per
3 10,000. As far as cigarette smoking is concerned, a
4 risk of one in ten thousand would result from smoking
5 about one cigarette every six months.

6 MS. HEBERT: Could we talk about reproductive
7 problems and liver damage.

8 MR. ZAR: Would you mind waiting until we
9 finish with this one question, please.

10 MS. HEBERT: Sure, go ahead, finish, please.

11 MR. ZAR: Do you have more to say?

12 DR. NISBET: I think as far as answering that
13 specific question that's sufficient.

14 DR. OYEN: Thank you.

15 MR. ZAR: Are there other questions?

16 UNIDENTIFIED CITIZEN: I have one. A
17 comparison made on the up -- fish upstream from Dow
18 with the fish downstream from Dow.

19 MR. AMENDOLA: Yeah. As you may know there
20 is a -- or used to be a dam called the Dow dam right in
21 the middle of the Dow plant and that dam served
22 somewhat as a barrier to fish moving upstream, however,
23 it did have a fish ladder associated with it all the
24 time. There were data collected in, I think, 1978. In
25 1980 and 198 -- I think '78 and '80 were the two

1 principal times, and the fish collected downstream of
2 Dow's effluent clearly were much higher in
3 concentration than any of those found upstream. In
4 some cases some levels of dioxin were found in some of
5 the upstream fish, but not to the same extent. I trust
6 that answers your question.

7 MR. MOORE: Dr. Oyen had an article in the
8 paper here within the last ten days stating that radium
9 has been found in the deep water wells of Midland and
10 Saginaw and Bay Counties. Did you find any radium in
11 the Dow brine which is down 5,000 feet and if so what
12 hazard would that pose?

13 MR. ZAR: Can you answer that?

14 MR. AMENDOLA: No.

15 MR. ZAR: I don't think we looked for
16 radioactive materials in this study and if Dr. Oyen
17 would like to respond as she was there.

18 DR. OYEN: Well, I don't know anything about
19 the analysis of Dow brine at 5,000 feet, these are 500
20 -- 400 foot wells with naturally occurring radium 226
21 and 228. It's a coincidental finding but we're
22 following up on it.

23 MR. MOORE: The paper said 1,000 feet.

24 (Inaudible comments.)

25 MR. MOORE: Yes, it did.

1 MR. ZAR: Would the gentleman who asked the
2 radium question please state his name. Would you state
3 your name, sir?

4 MR. MOORE: Moore, Albert Moore, Ingersoll
5 Township.

6 MR. ZAR: Thank you. Any other questions?
7 This gentleman.

8 MR. PALUM: Yes, my name is John Palum, and I
9 have a question about pica. Pica has been referred to
10 here as an unusual occurrence, an uncommon medical
11 condition. I could be wrong, but it's my understanding
12 that it's really not all that uncommon. Can you verify
13 that for me.

14 MR. ZAR: Dr. Nisbet.

15 DR. NISBET: I don't remember exactly in what
16 words I characterized it in. It is not a rare
17 phenomena, depending on how it's defined, it occurs in
18 something like 1 to 3 percent of children.

19 MR. ZAR: Do you have an additional comment
20 on that, Dr. McClanahan? Dr. Johnson?

21 DR. JOHNSON: I think I would state that it
22 is a rare occurrence, classical pica. Now, every child
23 goes through a phase of putting things in his mouth and
24 that's not what we're talking about. This is really an
25 excessive amount. And perhaps the 1 to 3 is reasonable

1 there but it only lasts between the ages of about one
2 and a half and three.

3 MR. PALUM: So, do you call it pica when
4 it's --

5 DR. JOHNSON: When it continues or gets
6 beyond that, yes.

7 MR. PALUM: Beyond that between one and a
8 half to three. (Inaudible comments.)

9 DR. JOHNSON: Well, that's normal behavior
10 but I'm talking about really excessive. (Inaudible
11 comments..)

12 MR. ZAR: Dr. Barnes has a comment on that.

13 DR. BARNES: Just to say that something which
14 seemed as common as kids getting their hands dirty and
15 putting them in their mouth, I was surprised to find
16 about five years ago that there was really very little
17 hard data on that. And much of what you have are
18 people who are experts in this area who have started
19 giving their professional judgment. Over the past
20 three or four years, however, a series of studies have
21 been conducted in this country and elsewhere trying to
22 get a handle on that question, how much does the
23 ordinary child eat, and those data are now coming in.
24 Dr. Nisbet can comment on the risks.

25 DR. NISBET: Well, Dr. Barnes has said half

1 of what I was going to say. The estimates of the,
2 quote, normal, unquote, intake by children that is used
3 in the risk assessment are based on the recent studies
4 that Dr. Barnes referred to and those are in the range
5 of 100 to 500 milligrams swallowed per day. Depending
6 on how pica is defined that may involve intakes in the
7 amount of 10 grams or even more of soil per day.

8 MR. ZAR: Any further questions? The
9 gentleman back there.

10 MR. PINE: My name's Harry Pine, I'm a
11 resident of Midland. I just wanted to ask them about
12 your recommendation that Dow monitor the sediment in
13 the river. Do you have any idea what level you would
14 regard as acceptable of dioxin in the river and do you
15 have any ideas as to what type of things could be done
16 about the sediment?

17 MR. AMENDOLA: Thank you, that's a very good
18 question. In response to the first part of your
19 question about what level would be acceptable, right
20 now there are no sediment criteria per se as to
21 classification of river or lake or harbor sediments for
22 safe levels of dioxin. The concern we have about the
23 sediments is that there may be levels of dioxin in
24 pockets in the river where organic material may have
25 accumulated or may have been exposed because of the

1 flood that might be introducing fairly significant
2 quantities into the river. We don't think it is a very
3 likely event, however, it's something that we believe
4 should be looked at.

5 What we had in mind, and is pointed out in the
6 draft risk management report, was some sort of a
7 classification system to evaluate the sediments either
8 visually or with some gross measure of organic analysis
9 and then following up those that appear to be abnormal
10 from the general river with a dioxin analysis. In the
11 case of the Tittabawassee River our past sediment work
12 has shown we have not been able to detect 2378-TCDD
13 directly. We found at detection levels of 10 to 30
14 parts per trillion, however, we have found higher
15 chlorinated dioxins and furans. The river, of course,
16 as the fishermen here might know, has got a kind of a
17 sandy gravelly bottom, which is not very conducive,
18 fortunately, toward collecting a lot of organic
19 materials which might contain dioxin. I hope that
20 responds to your question.

21 MR. ZAR: Further questions?

22 MR. MILLER: Yes. My name is Terry Miller,
23 I'm a resident of Bay County, and I was asking
24 questions around the sampling that was done on the
25 water intake in the bay, Saginaw Bay. When were those

1 samples taken?

2 MR. AMENDOLA: I believe those samples were
3 taken in 1984. I'd have to go back and check the exact
4 dates.

5 MR. MILLER: Have they proved to be
6 insignificant?

7 MR. AMENDOLA: No, none detected. We could
8 not measure 2378-TCDD down to a level of I believe it
9 was less than 5 or 10 parts per quadrillion, which is
10 the state of the art for analytical work in drinking
11 water supplies.

12 MR. MILLER: But as a follow-up question what
13 I'd like explained, if possible, how do you account for
14 the fact that in '85 there was a report based on the US
15 Fish and Wildlife Service in the adult common Tern, of
16 birds like the seagull, they found 25 parts per
17 trillion of dioxin and in Tern eggs 3763 parts per
18 trillion. And what this gentleman says is although
19 humans do not eat Terns, what is happening with them is
20 an environmental barometer for possible human health
21 effects, T. J. Miller, contaminant specialist with the
22 agency in the East Lansing office said on this date,
23 January 10th, 1985, the study supports Canadian data
24 that dioxin levels inherent in gull eggs from the
25 Saginaw River, mouth and bay, 85 parts per trillion are

1 in the highest in the Great Lakes.

2 MR. AMENDOLA: We are aware of those data and
3 it's not surprising to us that there is some
4 contamination or accumulation of these pollutants in
5 the food chain. In many cases the birds eat a lot of
6 fish, which have low levels of contamination in them
7 and dioxin is bio-accumulative. One of the unfortunate
8 things about this chemical is that it doesn't degrade
9 very readily in the environment and levels that -- or
10 discharges that may have occurred several years ago may
11 still be having their effect. I think Dr. Nisbet would
12 like also to respond.

13 DR. NISBET: I can respond to that because I
14 also study Terns. They are very -- as predecessors who
15 are at the top of the aquatic food chain they are very
16 efficient at concentrating chemicals such as dioxin and
17 it's not unusual to have concentration factors of a
18 hundred thousand or a million between water and the
19 common Tern eggs. So there is no inconsistency between
20 finding tens of parts per trillion in the Tern egg and
21 not finding dioxin in the water at less than one part
22 per quadrillion.

23 MR. MILLER: If I may ask another question.
24 The risk advisory, there's been a lot of emphasis about
25 the PCB content, of course, that's why we're here this

1 evening. But in the study you indicated that seven out
2 of the nine chemicals found in fish aren't probable
3 carcinogens but are known carcinogens. Were those
4 carcinogens factored into the risk analysis when it
5 came to the fish advisories?

6 MR. ZAR: I think -- Are you asking about the
7 fish advisory?

8 MR. MILLER: Right, now on the Tittabawassee
9 fish. I mean there were a number of chemicals that
10 were found during your analysis of those fish. Now,
11 the ones that you're looking at --

12 MR. ZAR: Are you asking -- You're asking
13 whether it was figured into the Michigan fish advisory.
14 And Michigan would have to answer a question like that.
15 Is that the question?

16 MR. MILLER: I guess I was asking whether the
17 EPA --

18 MR. ZAR: Can I put you on the spot, John?

19 MR. HESSE: Sure.

20 MR. ZAR: I already did, I guess.

21 MR. HESSE: We're aware of a lot of chemicals
22 -- a lot of chemicals can be found in fish at low
23 levels and not just in the Great Lakes or just the
24 Tittabawassee River, but it's fairly common. I think
25 IJC has identified something like a thousand chemicals

in the Great Lake system, sediment, water, fish, and a lot of them have shown up in fish. There's no real easy way to predict what the accumulative effect of all of them would be in combination. Some studies show that they're protective of one another or act antagonistically, some may look like they could be synergistic. PBB and PCB, for example, some studies have been done recently show that one -- the presence of the two of them protect against the effect of the other. So we at this point do take it into consideration, the levels of the other contaminants. The level in the Walleye PCBs in the Tittabawassee River appears to be just about the same level as what we're seeing in the Walleye in Lake Erie and Lake St. Clair and just about anywhere we collect, about four tenths of a part per million on an average. And it's -- And we use a sort of a protective way of taking that into consideration in that when 10 percent of the fish exceed any one of the FDA standards we do put a restrictive advisory on it. In this case the Walleye are not -- none of them are exceeding the standard for PCB, for instance. None of them are exceeding -- we don't have an official standard for dioxin but they're not exceeding the 10 parts per trillion trigger that we use. So it's just -- it's that state of the art that

1 we're using at this point. Those fish -- We're
2 applying the same process to these fish as what we're
3 trying to do in the other waters we study. It's
4 nothing real unique in this river.

5 MRS. MANION: You're saying you
6 consider --

7 MR. HESSE: Well, we don't consider, we've
8 seen the data. I'm not sure about seven and nine.
9 They're not added yet. We're aware of the presence of
10 the chemicals, I don't know about the seven and nine
11 being known carcinogens.

12 MRS. MANION: That's what it says on the
13 report.

14 MR. HESSE: I haven't read that last bit of
15 detail in that report.

16 MRS. MANION: It also says the PCBs
17 (inaudible comments).

18 MR. ZAR: Excuse me, miss, could you speak
19 up?

20 MRS. MANION: It says for PCB the upper bound
21 of cancer risk by the consumption risk of Walleye from
22 the Tittabawassee River similar to those posed by the
23 PCB and the CDDS.

24 MR. HESSE: If we apply the FDA or the EPA
25 risk assessment to fish in general, our EPA method, and

1 compare what the risk would be, even fish that meet the
2 FDA standards, you're aware of the two parts per
3 million FDA standards for PCBs, if we apply the same
4 risk assessment approach that FDA is applying, fish
5 meeting the FDA standard and, therefore, legal for
6 human consumption -- or sale for human consumption
7 carries a risk of approximately four in a thousand.
8 That -- Each one of the FDA standards carry -- if the
9 fish are right at the level carry about that risk using
10 that type of methodology. None of the fish in the
11 Great Lakes would meet the one in the hundred thousand
12 level if you use that kind of methodology.

13 We have to consider some of these other mitigating
14 factors when we take into consideration advisories.
15 It's not just the Great Lakes that would have problems
16 if we used -- applied that directive without
17 considering some of these other factors that I
18 mentioned earlier. Our Perch in Lake Michigan, for
19 instance, have very, very low levels, ten parts per
20 billion, of PCBs and yet they would carry a risk of
21 greater than the one in a hundred thousand estimated
22 risk of cancer. Yet, that's a very common figure in
23 fish anywhere in the United States. I don't know if
24 very many fish would have less than that.

25 MRS. MANION: Give me the bottom line,

1 though, are you doing one chemical at a time?

2 MR. HESSE: Yeah, one chemical at a time.

3 MRS. MANION: You didn't take into the
4 account the other nine when you did the --

5 MR. HESSE: Only in the process of applying
6 the 10 percent rule, and they would be added in that
7 way. If they -- If one fish out of ten had PCBs,
8 another fish had chlordane, then they would exceed the
9 10 percent rule and we would put a restriction on that
10 fish on that basis. Whereas they would be perfectly
11 fit for eating -- for sale in the commercial market.
12 But the average on any of them wouldn't come anywhere
13 near the FDA standards.

14 MRS. MANION: Are you saying you know or you
15 don't know the risk of these others?

16 MR. HESSE: I don't think we know the risk on
17 any of them, the actual true risk. It may be several
18 orders of magnitude what the difference in the true
19 risk in terms of what we projected. But it allows us
20 to compare the fish from the Tittabawassee from fish
21 elsewhere. And what I'm saying is that Tittabawassee
22 River fish aren't that much different than other fish
23 in other waters when we look at the comparable levels.

24 MR. MILLER: So other fish in the Great Lakes
25 we expect to find similar values of these kinds of

1 chemicals?

2 MR. HESSE: Read them off for me.

3 MR. MILLER: PCTs, PCBs, chlordane, DDT,

4 yeldren, exchlorabenzine, atropine, atrachlorastylene,

5 petrachloratoxide.

6 DR. NISBET: Yes.

7 MR. HESSE: Yes, you'll find those same

8 chemicals in Lake Michigan fish.

9 MR. MILLER: And with pretty much the same

10 values in terms of content?

11 MR. HESSE: I believe so. We did the

12 analyses on those, I'm sure if that's the '85 data that

13 you put in there, yes.

14 MR. ZAR: Can I move on to a different item.

15 It sounds like you have some detailed interests there

16 that are perhaps pursued directly at some other time.

17 If I could, I'd like to take one or two more

18 questions, move onto statements and then if we have

19 some time at the end take some more questions. That

20 gentleman.

21 MR. MULLISON: My name is Wendal Mullison,

22 I'm a resident of Midland. I want to ask Dr. Nisbet a

23 question. In using the data for the toxic effects of

24 2378-TCDD in the fish, was it the whole fish, analysis

25 of the whole fish or was it an analysis of the flesh

1 eating part of the fish that people would eat that you
2 used in making your risk assessment?

3 DR. NISBET: The exposure assessment was
4 based specifically on fillets and we combined data for
5 fillets with skin on and fillets with skin off. Where
6 they compared there was not a substantial difference.
7 So we pooled all the data for fillets.

8 MR. MULLISON: Yes. My question, though, was
9 the analysis of the content in the fish, was that based
10 on analysis of the whole fish or was it based upon
11 separate portions of the fish?

12 DR. NISBET: No, it was based on the edible
13 tissue only, only on the fillets. There was some other
14 analyses where the whole fish were analyzed and we
15 didn't use those as part of the exposure assessment
16 because those concentrations are likely to be higher
17 because they are -- dioxin is concentrated in the
18 viscera.

19 MR. ZAR: This gentleman.

20 MR. MARTIN: Yeah, my name is Doug Mark, I've
21 got a question for Dr. Nisbet. I just wondered if you
22 had ever studied the correlation between dioxin and
23 maybe the possibility of smoking in lieu of lung cancer
24 in respect to the dioxin that's been found in the
25 quality of paper with using the chlorine process with

1 your dioxins in your paper, your cigarette paper, and
2 also if you chew tobacco and get lung cancer, you don't
3 have to light it up. I just wondered about the residue
4 on the tobacco, have they ever looked at this as maybe
5 being a major possibility of lung cancer?

6 DR. NISBET: I don't know of any studies
7 which have investigated dioxin levels in either
8 cigarettes or in tobacco smoke concentrate. It's only
9 very recently that some have been found in papers, in
10 cigarette papers, and that investigation hasn't gone on
11 very far yet.

12 MR. ZAR: One last question.

13 MRS. MANION: I have two questions. When you
14 do air sampling are there recovery rates for soil and
15 water?

16 MR. AMENDOLA: There are two issues in air
17 sampling dealing with -- dealing with that question.
18 The first is capture efficiency, and that is a measure
19 of whether or not the device you're using to collect
20 the air sample is catching everything that's in that
21 air sample. For both incinerator emission testing and
22 ambient air testing for dioxins there is some
23 uncertainty as to whether the capture devices,
24 collection devices are catching 100 percent of all the
25 material. There have been some recent studies, and

perhaps Dr. Barnes, do you have some comments on those.

DR. BARNES: Just on the question?

MR. AMENDOLA: Yes.

DR. BARNES: There was a study done for the Environmental Protection Agency to investigate the emissions of dioxins from combustion sources and some average results were obtained that raised that question about this, how good the capture efficiency was. And I must say I lost track of how that turned out. This was some time ago now, but people are going back to take a look to see what the answer to the question was. As I've just asked casually about it, I can give you some more detailed information when I get back to my office. There were questions raised about that study that the people had originally raised questions about it but now they think maybe it was not a problem. But to answer your question honestly, it's unsettled in my mind and I'd have to go back and check to see what the most recent information is.

MR. AMENDOLA: I would just like to point out that in the testing that was done here we did use what was thought to be the best state of the art for the air analysis for sampling technology. In the case of the ambient air samples we used a two phase system. The first was a fiber filter, which was commonly used to

1 collect particulate matter in air, and that was backed
2 up by a polyurethane foam cartridge and all the air
3 that went through the filter then went through this
4 cartridge. And what we did find as part of that work
5 was that some of the higher chlorinated dioxins, more
6 of those were captured on the filter, and some of the
7 tetra through pentadioxins, more of those were captured
8 on the polyurethane foam. And that leads us to believe
9 that we had a pretty good collection efficiency,
10 although, you know, I can't state that it was 100
11 percent.

12 The second part of your -- of the issue of
13 recovery that you raised is one dealing with analysis.
14 And the analysis of an air sample, once the residue is
15 collected it's subjected to essentially the same types
16 of extraction and extract clean-up, analytical
17 techniques as the soil or fish or any other type
18 sample. There are recoveries associated with that.

19 In the air study we initially established some
20 very stringent percent recoveries on the analytical
21 systems and later we found that we didn't achieve all
22 of those, but by and large the data obtained fell
23 within acceptable ranges for recovery.

24 MRS. MANION: Does the recovery rate change
25 the, you know, like the level of parts per billion for

1 recovery?

2 MR. AMENDOLA: Well, in the analyses the
3 final result you get is adjusted for the recovery rate
4 of a surrogate compound or spike level compound, so you
5 take that into account in reporting the results.

6 MRS. MANION: So what was the -- I don't
7 think you listed in the report like even a range of
8 your -- (inaudible comments)

9 MR. AMENDOLA: In the risk assessment report
10 I'm not -- we do have a companion report that has more
11 detailed work on air, I'll be happy to get you a copy.
12 And it'll have all those recovery rates specified for
13 each sample that was collected and analyzed.

14 UNIDENTIFIED CITIZEN: The second part of my
15 question is why do you use double negatives when you
16 express the conclusions of your work?

17 MR. AMENDOLA: You're talking about "does not
18 pose unacceptable risks." I think that's kind of a
19 term of art in risk communication that has evolved, and
20 I don't know the origin of it, perhaps some of the
21 other fellows on the panel do. Dr. McClanahan, do you
22 want to touch that one?

23 DR. McCLANAHAN: I'm not in charge of public
24 records.

25 MR. ZAR: Are there any burning questions?

1 I'll take one burning question only and then we have to
2 give people a chance to make statements. This lady has
3 a burning question.

4 BARBARA: My name is Barbara and I'm from the
5 Department of Natural Resources in Ann Arbor. I have a
6 question for EPA about how you handle the other
7 chemicals, the PCBs and other chemicals that were in
8 the fish, how do those impact on the risk assessment,
9 both carcinogenic and non-carcinogenic?

10 DR. NISBET: Yes, that's addressed
11 specifically in Appendix B to the report. The -- Both
12 the carcinogenic risks and potential non-carcinogenic
13 risks posed by these nine chemicals are considered
14 briefly in that appendix.

15 BARBARA: Yes, but you did not say how they
16 impacted on your risks of dioxin.

17 DR. NISBET: The -- Briefly eight of the nine
18 compounds or groups of compounds posed completely
19 negligible risks relative to the dioxin. The PCBs
20 would pose risk both cancer and non-cancer effects,
21 which would be in the same ballpark as the dioxins. So
22 the two together would be on the order of twice the
23 risks calculated for the dioxins alone if the effects
24 were additive. Now, we discussed whether they might be
25 more additive or more than additive or less than

1 additive and the evidence one way or the other is very
2 scanty, so we didn't draw any conclusions in that
3 regard.

4 MS. HEBERT: She's not just talking about
5 PCBs and dioxins, she's talking about other chemicals.
6 You haven't addressed that.

7 DR. NISBET: I specifically addressed all the
8 chemicals which have been detected in the fish.

9 MS. HEBERT: So list them, please. Would you
10 please list them.

11 MR. ZAR: There are nine chemicals listed in
12 the appendix which --

13 DR. NISBET: Appendix B.

14 MR. ZAR: Appendix B.

15 MS. HEBERT: So what are they?

16 MR. ZAR: And they were read into the record
17 earlier.

18 MS. HEBERT: Why don't you just list them
19 now, Howard?

20 MR. ZAR: I said I wouldn't.

21 MS. HEBERT: You wouldn't what?

22 DR. NISBET: Would you like to take public
23 comments now?

24 MR. ZAR: Yeah, I'd like to take public
25 comments. You asked quite a few questions, miss.

1 MRS. MANION: It's just one quick one. On
2 your map on page 60 you have dots and circles, would
3 you just explain what those each signify as to the
4 sampling?

5 MR. ZAR: Double negatives, now dots and
6 circles and then we'll take comments.

7 MR. AMENDOLA: On page 60 this is a graph or
8 a chart that I had nothing to do with. I believe the
9 dots, the ones that are filled in, are samples that
10 were collected and analyzed and the circles were
11 samples that were collected but not analyzed. As you
12 recall at the time we did this oil study we took more
13 samples than we analyzed in case we had to go back to
14 reinforce or assure ourselves of what our conclusions
15 were.

16 MS. HEBERT: Can you take --

17 MR. AMENDOLA: Excuse me, I'd like to finish
18 this answer, please.

19 MS. HEBERT: Go ahead.

20 MR. AMENDOLA: So we didn't take -- we didn't
21 analyze every sample collected. And the circles
22 represent those that had not been analyzed.

23 MR. ZAR: That will be the end of the
24 question period. If we have some time at the end we
25 can take some more questions. Are there any

1 individuals who wish to make statements? This
2 gentleman.

3 MR. MILLER: Yes, Terry Miller from Bay
4 County. I hadn't originally intended to make this
5 statement but a member of the Michigan Department of
6 Health brought up Barry Commoner and apparently he had
7 addressed the EPA earlier in the year and I think just
8 a few of his comments might be appropriate this evening
9 and then I would like to finish with a comment of my
10 own.

11 Our environmental legislation, and this comes from
12 a presentation by Dr. Barry Commoner, our environmental
13 legislation ignores the origin of the assaults on
14 environmental quality, fails to recognize that
15 environmental pollution is an essentially incurable
16 disease that can only be prevented and instead deals
17 with its symptoms.

18 The present largely unsuccessful regulatory effort
19 is based upon the now well established procedure.
20 First the EPA estimates the degree of harm caused by
21 different levels of various environmental pollutants.
22 Next some acceptable level of harm is chosen, for
23 example, a cancer risk of one in a million. And the
24 EPA establishes emission standards that can presumably
25 achieve that risk level.

1 Polluters are then expected to introduce controls
2 such as auto exhaust catalysts or power plant stack
3 scrubbers, read sand pits or sand traps that will lower
4 emissions to the required level. If the regulation
5 survives the inevitable challenges from industry and in
6 recent years from the administration itself the
7 polluters will invest in the appropriate control
8 systems. If all goes well, and it frequently does not,
9 at least some areas of the country and some production
10 facilities will then be in compliance.

11 Clearly this process is the inverse of our
12 preventive approach to public health. It strives not
13 for a continuous improvement in environmental quality
14 but for the social acceptance of some presumably low
15 risks to health in a way that represents a return to
16 the medieval approach to disease in which illness and
17 death itself was regarded as a devil on life endured as
18 payment for original sin. In our updated version we
19 think that some level of pollution and some risk to
20 health is the inevitable price to be paid for the
21 material benefits of modern technology. Some of us are
22 not willing to accept that.

23 And many of us feel, and this is from my position,
24 this isn't Dr. Commoner, many of us here, many of us
25 will feel far more comfortable when the Dow Chemical

1 Company eliminates product lines that produces and
2 introduces dioxin into the environment regardless of
3 how small the quality. Thank you.

4 MR. ZAR: Any further comments on the risk
5 management or risk assessment reports?

6 MR. HEBERT: Yes, Howard, I'll see you in
7 court about that sample at Rockwell. Yes.

8 MR. ZAR: It's a statement, I guess. Next
9 comment. Gentleman in the green jacket.

10 MR. KUTCHIN: Yes, I've been sitting here
11 listening --

12 MR. ZAR: Your name, sir.

13 MR. KUTCHIN: The name is Sam Kutchin of
14 Midland here, but as Mayor pro tem McCaffrey thanked
15 you all for being here I think we've got to take into
16 consideration that a lot of these people here aren't
17 sitting home and watching the boob tube, they're out
18 here, you know, trying to get informed about things and
19 I think it would be nice, since their names are up
20 there, if we could get a summary of the report of
21 tonight's meeting.

22 MR. ZAR: You're asking for a summary of the
23 meeting, of this meeting?

24 MR. KUTCHIN: Yup.

25 MR. ZAR: I think we can do that. We intend

1 to, I guess I didn't say this, but we intend to put
2 into the repository a summary of the comments received,
3 not only of those made tonight but also those received
4 in writing, as soon as we get it done and also
5 certainly the risk -- the final risk management
6 document when we get it done. The locations of the
7 repositories are listed on the fact sheet, the gray
8 document that we held up a few times.

9 MR. KUTCHIN: Thank you.

10 MR. ZAR: Any other statements? The
11 gentleman in the back.

12 MR. FAREEVY: Yeah, I was just wondering, I
13 heard chlorine mentioned a couple times along with
14 dioxin. In an offbeat way is there any relation
15 between dioxin and chlorine?

16 MR. ZAR: Fluorine?

17 UNIDENTIFIED CITIZEN: Chlorine.

18 MR. ZAR: Chlorine?

19 MR. FAREEVY: Chlorine, yeah.

20 DR. BARNES: Dioxin, the way it's been
21 defined here tonight, is a -- it is a compound which
22 contains chlorine. Table salt contains chlorine, a lot
23 of things contain chlorine. But particular -- So,
24 there's nothing inherently bad about chlorine itself,
25 but in a particular combination with other atoms it can

1 form compounds which are problems, and 2378-TCD, which
2 we're saying is dioxin, is such a problem -- is such a
3 compound that can produce a problem.

4 I'm sorry, we didn't get your name for the record.

5 MR. FAREEVY: Jack Fareevy.

6 MR. ZAR: The gentleman in the brown jacket.

7 Your name, sir?

8 MR. MOORE: I'm Pat Moore from Ingersoll, and
9 I've been in on this pollution bit since about 1970
10 when the Ingersoll Township Zoning Board first banned
11 the deep well injection of Dow chemicals in the
12 Township of Ingersoll. Ingersoll Township Board has
13 been in on this right from the start. And when those
14 words, dioxin, came out they saw it then as information
15 that they had not been able to get before. So right
16 from the beginning Ingersoll has been in favor of the
17 EPA coming in here.

18 I attended the first meeting down in the union
19 hall that Don Albosta called, Dave Stringham of the EPA
20 Chicago carried the load. Gary Amendola was not even
21 in on it at that time. Later on Amendola came into the
22 picture. I was really surprised tonight to hear the
23 words of Harry McCaffrey. I remember some of the words
24 that he and Mann and Bill Welch spoke when they were
25 coming in here. They were very, very unhappy. We were

1 going to be Times Beach, we were going to be Love
2 Canal, we were going to have our image ruined. The
3 money changers on Main Street were no longer going to
4 hear any clinking in their cash registers. Was a
5 horrible thing to have these guys come in. They come
6 in, they did their job, and they're giving their honest
7 opinion, and I think it's a wonderful thing that they
8 did come in.

9 Now, if we have to look back to see where we were
10 say ten years ago to see how far we've come, let's take
11 a look at a few of the things as they were ten years
12 ago. Let's go to Bay County, there's been a benzene
13 spill near Auburn that was allowed to soak away,
14 nothing done. DNR didn't do anything. After we got
15 these people activated and the DNR moving it was
16 cleaned up. It was found that instead of dissipating
17 and biodegrading as Dow had told them it would it was
18 scattered over a wide area, and I understand it cost
19 about a million dollars out there to try to clean that
20 up.

21 Along in the '50s put a lot of benzene tars along
22 Rockwell Drive in Bay County. It wasn't until the late
23 '70s that a lot of the people out there was wondering
24 what it was all about. One hunter went in there with
25 his dog, he sat down, he went home and he lost all the

1 hair off his butt. Another dog made the mistake of
2 lapping it up, his life was short, he was gone the next
3 day. Stan Wasic, who was supervisor of Midland or
4 Williams Township heard about some of this stuff, he
5 went over there and was wandering around there to see
6 what it was all about. Dow security caught him in
7 there and they were going it put old Stan in the
8 hoosegow. He had to do a lot of fast talking to tell
9 them who he was and so on to get out of there.

10 At the last meeting we had at Williams Township
11 Hall in which the Dow officials attended they invited
12 all of us, including Stan Wasic, to go out and look at
13 this new Rockwell -- new Rockwell, the songbird
14 landfill which is supposed to be the Cadillac of all
15 landfills. The Rockwell landfill is now kept, it is
16 now monitored and there's no more of that seeping down
17 the drain. The Poseyville landfill was never supposed
18 to have any toxic substances in it. Any of you drive
19 out Poseyville Road know that that is now monitored, it
20 is now drained and it is kept. The deep well injection
21 of toxic substances has ceased.

22 Under the City of Midland all Dow property being
23 injected to both toxic substances they have, the Dow
24 Chemical under ground, under pressure. When the
25 Fortune magazine editor was in here he asked one of the

1 people from Dow Chemical where that stuff was going
2 after it was injected in there, they said they didn't
3 know. I want to add that they didn't give a damn.
4 They also injected the brine lace out in our
5 countryside and as the result of that we got a very
6 unusual smell in our water supply. Sandy Mannion can
7 tell you about that, it smells like rotten eggs, H₂S.
8 We asked Dow whether or not they were putting any
9 sulfur compound down there, they said no we're not
10 putting that. But what if you crack the rock structure
11 when you put this pressure off, and if you injected it
12 at a thousand pounds per square inch of the surface by
13 the time it gets down 5,000 feet you got enough
14 pressure to crack rocks as Dutch Boyle found out when
15 he ran the experiment way back in the '30s. Somebody
16 ask Dutch where that stuff was going that he was
17 cracking the rocks was going, he said, oh, I think that
18 may end up in Saginaw Bay. He was apparently working
19 in the Saginaw structure.

20 Now, that this is ceased my wife silver's no
21 longer blackens, you can go into the bathroom or the
22 kitchen without smelling rotten eggs. I don't know
23 about Mannion, yours gone, too?

24 MS. MANNION: Pretty much.

25 MR. MOORE: Yup. So, Dow didn't do it, it

1 wasn't Dow's stuff. But if you crack the rock
2 structure down there, release some of this stuff
3 there's only one place for the gas to come. And when
4 it hits your ground water supply you're going to have
5 it.

6 Now, we got other side effects from this. The
7 fact that EPA come in here and did what they did and
8 got the DNR started under Dr. Tanner, we could never
9 get Dr. Tanner up here, he never did anything. But
10 after the EPA came in maybe they had good graces or
11 maybe the other guys decided to enforce the law, the
12 oil field wastes are being taken care of. If any of
13 you have driven down through Porter, gone down to the
14 oil fields, you'll see areas there barren but salt
15 crystals on the ground from the oil field waste. If
16 you go through Williams Township you won't see that and
17 if there is a leak in one of those pits out there the
18 DNR is out there the next day.

19 The roadside dumping is practically seen. The
20 roadside dumping is practically seen. Not only that
21 but we're getting fast action. About a month ago there
22 was a diesel spill that got down the river, went
23 through the Dow property on the well. The fisherman
24 reported it. It wouldn't have happened ten years ago,
25 number one there wouldn't have been any fishermen in

1 there, if he had been he wouldn't have reported it
2 because he figured it come from the Dow property and in
3 Midland you do not report things about Dow if you want
4 to live in peace with your neighbors, I can tell you
5 that. The DNR was in there the next day. Not only
6 that but the TV stations were in, they were taking a
7 picture of the man, trying to put his hands over the
8 camera. He was hauled into court and fined. Not only
9 that but they caught him for having stored salt on his
10 property without proper coverage. And in the paper
11 tonight it said he got fined \$800 for that.

12 Now, this is a far cry from what we used to get
13 when we called Dr. Tanner. So I think the whole thing
14 has been very beneficial from everybody's standpoint
15 and I think from the standpoint of Dow it has been also
16 very beneficial. They spent something like 60 million
17 dollars enhancing their image. They didn't like the
18 Dow title with dioxin in it. They didn't like the
19 cartoonist. They didn't like the article of Wall
20 Street Journal, they didn't like it in Fortune, they
21 didn't like it in Business Week, it was destroying
22 their image. Popov, who's now head of the Dow
23 Chemical, made a speech recently in Sarnia and he said
24 that perception is reality. No matter what you tell
25 these people what they see is what they're going to

1 believe. And that is what happened in this case.

2 I thank you.

3 MR. ZAR: Thank you. I might mention that
4 back in 1983 when Howard Tanner wrote the letter that
5 got all this stuff started, and there were some other
6 things that got them started, too, but that certainly
7 was one of the items. This gentleman.

8 MR. RIO: My name's Mike Rio and I am the
9 manager in engineering and environmental and computers
10 for the Midland region of Dow Chemical and I would like
11 to thank EPA for this very comprehensive and exhaustive
12 study. I'd also like to thank Mr. Moore and Mrs.
13 Mannion and the folks who have firmly urged us through
14 the years to change the way we do our business, and
15 they certainly have had an impact on us and I think
16 it's been a positive impact because I think we would
17 all admit that today Midland is a very much cleaner and
18 better community than it has been in the past.

19 I'd also like to say that I think the study that
20 you completed and we've cooperated with you in both the
21 stages have been a very positive experience for me and
22 for the Dow Chemical Company. It's probably as
23 comprehensive and as substantive and exhaustive a study
24 that has ever been done on any community that I'm aware
25 of. And I think this will probably serve as a model

1 for other studies that EPA would want to do in the
2 future.

3 I'd also like to pledge to you and to the
4 community that we will continue the efforts. The
5 improvements that you've showed on the screen are not
6 something that's a thing of the past, we're going to
7 continue our efforts to make these numbers even lower
8 and make Midland an even better community to live in.
9 So thank you very much and thank the community for its
10 indulgence over the last several years.

11 MR. ZAR: This might be a good time to
12 introduce some other EPA people who have been involved
13 in this. There may be some state people here, too, but
14 -- and I don't know their names so I apologize for
15 that. But Jon Barney who has done a lot of the
16 editorial work and has been the project manager for the
17 risk assessment, he's worked harder than most of us
18 have up here on the risk assessment I'd say over the
19 last five years. Carol Witt is working on the RCRA
20 permit, John Perrecone back there somewhere is our
21 public affairs officer. Lisa Dubois is a contractor,
22 Louise Fabinski is somewhere out there is from ASTDR.

23 Are there any more statements or comments, please?

24 (No response.)

25 MR. ZAR: Are there any more questions for

1 the panels or panel or for the speakers? This lady.
2 Your name, please?

3 MS. JAMES: Sandy James. Do you have a
4 trigger point for, you know, the soil sampling where
5 you would take action, for instance, like you did in
6 the (inaudible), I think it was one part per billion in
7 soil samples. Does that still hold or I would like to
8 know (inaudible comments)?

9 MR. ZAR: Dr. McClanahan, you want to try
10 that one?

11 UNIDENTIFIED PERSON: What's the question?

12 DR. McCLANAHAN: Her question dealt with is
13 there a trigger point for the concentration of dioxin
14 in soil. As the document that was developed for Times
15 Beach specified that that was a site specific
16 evaluation, same sort of thing would have to be dealt
17 with for any other particular location. And into the
18 conditions of that particular city, (inaudible) might
19 be developed. So I mean you couldn't just say if you
20 find one it's automatically going to be an action
21 level. It would have to be developed based on specific
22 situations of that community or that particular group
23 of samples.

24 MS. JAMES: So you have a different trigger
25 point for each area that you investigate?

1 DR. McCLANAHAN: Yeah, that's the way it was
2 intended and that's the way basically it's supposed to
3 be.

4 MS. JAMES: In Midland what would the guess
5 be?

6 DR. McCLANAHAN: Guesses don't work.

7 MS. JAMES: Well, how do you decide?

8 DR. McCLANAHAN: Basically you go through the
9 same sort of risk evaluation for Midland for the group
10 of samples that were collected in Midland with the
11 people who are living in the community where the
12 samples were found, what the distribution of the
13 contaminant is and taking into consideration state of
14 the art in terms of the potency factor of the dioxins
15 at the time the calculation was made, the soil
16 ingestion rate and things that -- things have changed
17 over the years since that first calculation was made.
18 That was five years ago, six years ago, things change.
19 So, again site specific factors. It might be one but
20 you can't just guess at what the number is going to be.
21 If it's something that's going to be applied in this
22 specific community or whatever community you're dealing
23 with.

24 MR. ZAR: Gentleman on the right here.

25 UNIDENTIFIED CITIZEN: What range was that?

1 Is there a range? I know you can't indicate a
2 particular number but would there be enough for a lower
3 limit?

4 MR. ZAR: I'm sure there's some upper limit.
5 I mean we wouldn't want to leave 100 parts per billion
6 around someplace, but it'd just have to be dealt with
7 on a, you know, the particular instance that we were
8 seeing in that particular location at the time that
9 that occurred. So probably would be -- might not be a
10 whole lot different than one, it might be five. I've
11 seen many (inaudible) reviewing the estimates.

12 Exposure of average values for a -- for an area
13 not just on one sample that happens to be in excess of
14 the particular number. We basically deal with what's
15 more or less average concentration. It's also --
16 there's a risk as to upper bound, upper bound number.

17 MR. ZAR: One or two more questions, if there
18 are any. Anyone? An unlimited supply. Your name.

19 MS. MANION: Sandy Mannion. When did you do
20 dusting from inside homes? It's recommended for the
21 future, why didn't you do them?

22 MR. AMENDOLA: Again a very good question.
23 When we started out with the odds of the study we tried
24 to estimate what we considered to be at that time the
25 major or principal exposure routes, which would be

1 consumption of fish, air, possibly drinking water, and
2 soils, of course. We thought about doing dust samples
3 but we deferred to see what we got on the first phase
4 of the work. Unfortunately, you know this process has
5 taken much longer than I think anybody would have liked
6 and it was one of those things that if we rolled back
7 the clock we probably would have went around and
8 collected some vacuum cleaner dust or something like
9 that to get some estimate of indoor exposure.

10 MR. ZAR: Any more questions? We have one
11 more.

12 UNIDENTIFIED CITIZEN: I'd just like to thank
13 you for your efforts and even though it did take five
14 years it sounds like they were very worthwhile and very
15 positive results and I have a sense that you probably
16 don't get the same kind of partnership in other
17 communities throughout this state and perhaps even in
18 the other states. Good luck in the future. Thank you
19 very much.

20 MR. ZAR: Mr. Amendola has the longest
21 period, he's going to respond to that.

22 MR. AMENDOLA: I started working on this
23 project before it became a project, actually back in
24 1978. And I must say that over the last five or six
25 years the cooperation we've got from the city, the

1 people in the community and Dow Chemical have been
2 marvelous. We have not -- We certainly had some heated
3 discussions and arguments at times but when push came
4 to shove the cooperation was there and I think people
5 were generally interested in finding out what the
6 bottom line was as opposed to trying to obscure the
7 process. And I'd like to just thank everybody for
8 that.

9 MR. ZAR: Gentleman in the jacket.

10 UNIDENTIFIED CITIZEN: One quick question for
11 Mr. Amendola. I was just wondering who peer reviewed
12 the whole report? I recall a report approximately ten
13 years ago that the EPA read a multinational peer review
14 report and we've seen a lot of flack over and I
15 wondered who peer reviewed --

16 MR. ZAR: There's a -- I'll give a partial
17 answer, Gary can answer it. In the risk assessment up
18 front you'll find an acknowledgment section listing a
19 large number of people who participated in the
20 development and who reviewed it including several
21 people from other federal agencies.

22 MR. AMENDOLA: There's no more to add. Mrs.
23 Manion.

24 MS. MANION: I'd like to know, I think you've
25 recommended that Dow do some Walleye sampling for the

1 spring of '88, during the spring run. Why did you
2 recommend the spring run when the fish have been out in
3 the bay through the winter? Am I correct, is that --
4 they come in the river in the spring, so why wouldn't
5 you sample --

6 MR. AMENDOLA: I think maybe John Hesse could
7 talk about the habits of the fish somewhat, but our
8 understanding is that those fish are exposed to the
9 mouth of the river and in the river for some time
10 before they actually run up the river. The spring run
11 is one type of set of samples that will be collected.
12 We'll also analyze in the summer some more resident
13 fish, the Carp and the Catfish and possibly some game
14 fish. But as far as a continuing program to better
15 characterize the fishery, it's not done as an attempt
16 to find fish that might be low, for instance.

17 MS. MANION: I just wondered why you did it
18 in the spring.

19 MR. AMENDOLA: The spring run fish are fish
20 that people are catching and eating a lot of so it's
21 important to characterize those fish. And data we got
22 in '85, for example, where there was spring run, summer
23 Walleye and so forth showed very little, if any,
24 statistically significant differences in those fish
25 from different types of year.

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MR. ZAR: Some more questions?

(No response.)

MR. ZAR: If not we'd like to thank you for coming. We've enjoyed our five years in Midland, we're looking forward to a shorter period of time to close this out and hopefully things are cleaning up fairly well so we won't have to be here to the same extent in the next five years. Thanks again.

(Proceedings concluded.)

1 STATE OF MICHIGAN)
2) SS.
3 COUNTY OF SAGINAW)

4 I, Kathy Brown, Shorthand Reporter, do
5 hereby certify that I recorded in shorthand the proceedings
6 had and testimony taken in the aforementioned proceedings
7 on the 28th day of April, 1988 in the City of Midland.

8 I further certify that the foregoing and
9 attached 98 typewritten pages or parts of pages constitute a
10 full, true, and correct transcript of my shorthand notes then
11 and there taken.

12
13 
14 Kathy Brown
15 Shorthand Reporter
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APPENDIX A

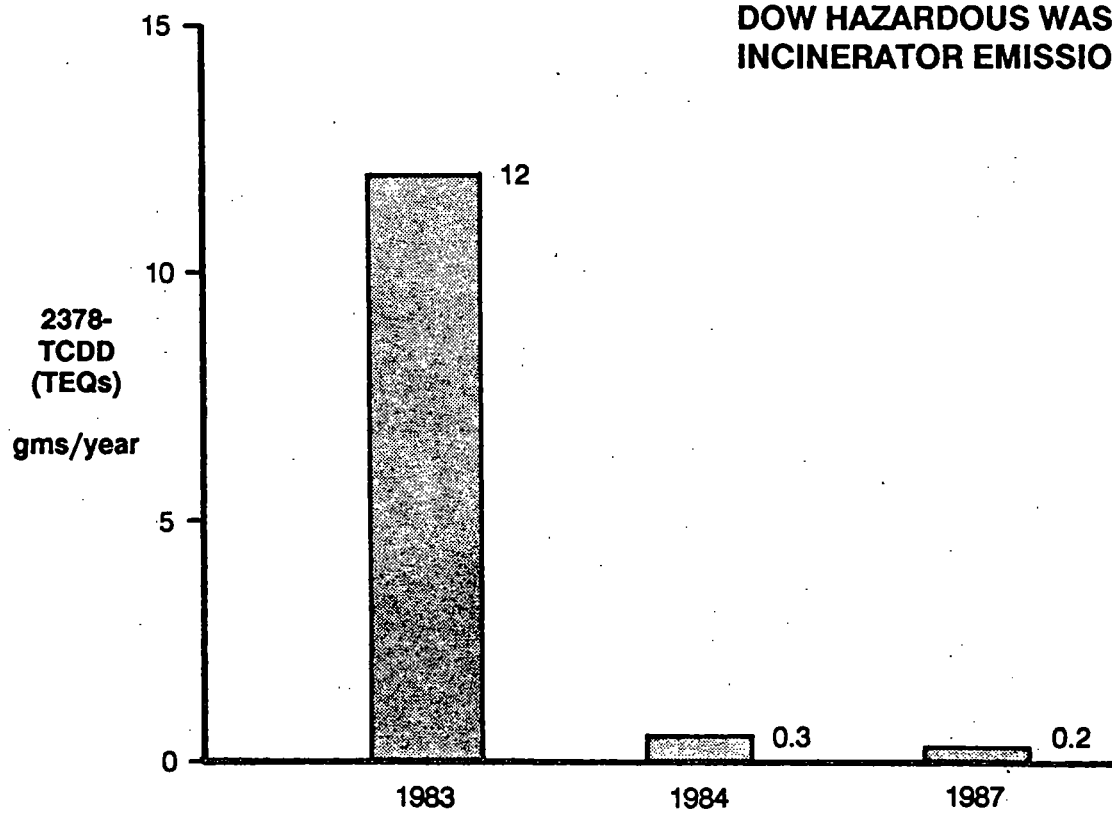
SITE HISTORY

- 1978 - Dow notifies MDNR of dioxin in fish**
- 1978 - MDPH issues Tittabawassee River fish advisory**
- 1978 to 1981 - MDNR/U.S. EPA studies**
- 1983 - State requests U.S. EPA assistance**

MICHIGAN DIOXIN STUDIES

- 1985 - City of Midland and Dow Soil Studies**
- 1985 - Drinking Water Studies**
- 1986 - Dow Wastewater and Tittabawassee River Studies**
- 1987 - Dow Incinerator and Ambient Air Studies**
- 1988 - Risk Assessment and Proposed Risk Management**

DOW HAZARDOUS WASTE INCINERATOR EMISSIONS



FISH CONTAMINATION CONCLUSIONS

- **Average levels in 1983 and 1985 fish are about the same**
- **Average levels in 1987 fish appear to be decreasing, but sample size is limited**
- **More studies are needed in 1988 and 1989 to confirm 1987 results**

INCINERATOR EMISSIONS AND AIR STUDY CONCLUSIONS

- **Incinerator emissions have decreased since 1983**
- **Dioxin was found in air outside Dow plant in 1983 and 1984**
- **Sources of dioxin contamination in Midland include current and past incinerator emissions, past process emissions, and windblown site dust**

DRINKING WATER STUDIES

- **2378-TCDD not found in water supply intakes from Saginaw Bay**
- **2378-TCDD not found in 16 drinking water wells tested outside Midland**
- **No other toxic organic chemicals were found in significant levels**

SOIL STUDY CONCLUSIONS

- **Highest 2378-TCDD levels found inside Dow plant; up to 36 ppb**
- **Contamination in Midland averaged <0.1 ppb 2378-TCDD**
- **Dow air emissions are likely source of contamination in city soils**

RECOMMENDED PRECAUTIONARY MEASURES TITTABAWASSEE RIVER FISH

- **Everyone should avoid eating Tittabawassee catfish and carp**
- **Women of child-bearing age and children should avoid eating any fish caught in the Tittabawassee River**
- **Others should limit eating Tittabawassee game fish to no more than one meal per month**
- **Any fish consumed should be cleaned according to MDPH recommendations**

PROPOSED RISK MANAGEMENT ACTIONS FOR DOW CHEMICAL

Wastewater

- **Research on Additional Treatment**
- **Incinerator and Pond Sediment Studies**

Air

- **Improve Incinerator Controls**
- **Dust Suppression Program**

RECOMMENDED PRECAUTIONARY MEASURES MIDLAND AREA SOILS

- **Parents of toddlers and children with pica should encourage children to keep soil out of their mouths**
- **Thoroughly wash hands after exposure to outdoor soil**
- **Wash or peel home-grown vegetables before eating**
- **Clean house to remove indoor dust**

PROPOSED MONITORING PROGRAMS FOR DOW CHEMICAL

- **Continued Wastewater Discharge Monitoring**
- **Supplemental Incinerator Emissions and Ambient Air Testing**
- **Continued Tittabawassee River Fish Monitoring**
- **Tittabawassee River Sediment Monitoring**
- **Limited Food Chain Studies**

EXPOSURE SCENARIOS

	<u>Higher</u>	<u>Lower</u>
AIR	Fenceline	Residential Area
SOIL	More Exposure Child with Pica	Less Exposure
FISH	Maximum Consumer High Sports Fisherman	Occasional Consumer Median Sports Fisherman

**SUMMARY OF ESTIMATED UPPER BOUND CANCER RISKS
FROM EXPOSURE TO DIOXIN CONTAMINATION
IN MIDLAND, MICHIGAN**

Estimated Upper Bound Cancer Risk		
<u>Exposure Route</u>	<u>Higher Estimate</u>	<u>Lower Estimate</u>
Fish	1 In 100 (maximum consumer)	1 In 1,000 (median fisherman)
	1 In 1,000 (maximum fisherman)	1 In 10,000 (occasional consumer)
Soil	1 In 100,000 (upper estimate)	1 In 1,000,000 (lower estimate)
	1 In 10,000 (child with pica)	-----
Air	1 In 10,000 (fenceline)	1 In 100,000 (residential area)

SUMMARY OF HAZARD INDICES FOR NONCANCER HEALTH EFFECTS

Exposure Route	Exposure Scenario	Hazard Index		
		<u>Long-Term</u>	<u>Short-Term</u>	<u>Single Meal</u>
FISH	Maximum Consumer	50	5	8
	Sports Fisherman - Avg.	9	0.7	0.2
	Occasional Consumer	0.7	0.4	0.2
SOIL	Upper Estimate Young Child			
	- with Pica	6	0.2	---
	- without Pica	0.6	<0.1	---
	Lower Estimate Young Child	<0.1	<0.1	---
	Upper Estimate Adult	<0.1	<0.1	---
AIR	Infant at Fenceline	3	0.1	---
	Child at Fenceline	1	<0.1	---
	Child at Residential Area	0.3	<0.1	---
	Adult in Residential Area	<0.1	<0.1	---

TOXICOLOGIC PARAMETERS FOR 2378-TCDD

Toxicologic End Point	Parameter Type	Parameter Value
Cancer	Dose-Response Slope (95% upper confidence limit)	1.6×10^{-4} (pg/kg/day) ⁻¹
Teratogenesis/ Reproductive Effects:		
long-term exposures	RfD	1 pg/kg/day
single-dose exposures	HA	300 pg/kg/day
Hepatotoxicity (liver effects):		
long-term exposures	RfD	1 pg/kg/day
short (10-day) exposures	HA	28 pg/kg/day
single-dose exposures	HA	280 pg/kg/day

**SUMMARY OF ESTIMATED UPPER BOUND CANCER RISKS
FROM EXPOSURE TO DIOXIN CONTAMINATION
IN MIDLAND, MICHIGAN**

Exposure Route	Estimated Upper Bound Cancer Risk	
	Higher Estimate	Lower Estimate
Fish	10^{-2} (maximum consumer) 10^{-3} (high sports fisherman)	10^{-3} (median sports fisherman) 10^{-4} (general consumer)
Soil	10^{-5} (upper estimate) 10^{-4} (child with pica)	10^{-6} (lower estimate) -----
Air	10^{-4} (fenceline)	10^{-5} (residential area)